STATISTICAL SECTION

# GUY'S HOSPITAL REPORTS.

EDITED BY
W. HALE WHITE, M.D.,
AND
W. H. A. JACOBSON, M.A., M.Ch.

VOL. XLIX,
BEING
VOL. XXXIV OF THE THIRD SERIES.

ARMY MEDICAL LIBRARY
Carvelles
7/v/45
WASHINGTON, D. C., U.S.A.
491033
29.4.49

LONDON:
J. & A. CHURCHILL, NEW BURLINGTON STREET.
MDCCCXCIII.

RC
31
L8G9
Ser. 3
v. 34

PRINTED BY ADLARD AND SON,
BARTHOLOMEW CLOSE, E.C., AND 20, HANOVER SQUARE, W.

# CONTENTS.


PAGE

In Memoriam—John Hilton, F.R.S. . xxxvii

A Biographical Sketch of Edward Cock, J.P., F.R.C.S. By R. Clement Lucas, B.S., F.R.C.S. xciii

I. The Prognosis of Ascites due to Alcoholic Cirrhosis of the Liver, to Perihepatitis, and to Chronic Peritonitis. By W. Hale White, M.D. . 1

II. The Pathology and Treatment of Puerperal Eclampsia, being a Portion of a Thesis for the Degree of M.D. in the University of Cambridge. By E. Petronell Manby, M.D., B.C., B.A. (communicated by A. L. Galabin, M.D. . 42

III. A Retrospect in Urethral Surgery. By C. H. Golding-Bird . . . . 71

IV. A Case of Homicidal Strangulation. By Thomas Stevenson, M.D. . . . 105

V. The Regeneration of Fibrous Tissue, as exemplified in the Union of Tendon. By A. H. Tubby, M.S. 109

VI. Some Cases of Malignant Disease of the Chest and Abdomen. By Frederick Taylor, M.D. . 123

VII. Note on the Operation of Circumcision. By N. Davies-Colley . . . . 163

VIII. On the Results of One Hundred and Thirty Cases of Excision of the Knee. By H. G. Howse, M.S. The Analysis of the Cases by G. Newton Pitt, M.D. 169

IX. Peritoneal Sanguineous Cysts and their Relation to Cysts of the Pancreas. By Theodore Fisher, M.D. (communicated by the Editors) . . 275

PAGE

X. Glycosuria and Surgery. By G. Bellingham Smith and Herbert E. Durham (communicated by the Editors) . . . . 335

XI. The Accidents of Anæsthesia. By George Rowell (communicated by the Editors) . . 433

XII. List of Specimens added to the Pathological Museum during the year 1892. By Lauriston E. Shaw, M.D. . . . . 467

List of Pupils who have passed the Examinations of the several Universities, Colleges, &c., 1892 . 485

List of Pupils who have received Appointments at Guy's Hospital, 1892 . . 491

# LIST OF ILLUSTRATIONS.


TO FACE PAGE

Autotype Portrait of John Hilton, F.R.S. . xxxvii

,, ,, Edward Cock, J.P., F.R.C.S. . xciii


## PLATES.


Mr. Golding-Bird.

Plates I—III illustrating his paper on Urethral Stricture 104


## PHOTO-MICROGRAPHS.


Mr. Tubby.

Illustrating his article on the Regeneration of Fibrous Tissue . . . . . 122


## WOODCUTS.


PAGE

Dr. Frederick Taylor.

Three Figures illustrating his paper on Some Cases of Malignant Disease of the Chest and Abdomen 159—161

Mr. N. Davies-Colley.

Three Figures illustrating his Note on the Operation of Circumcision . . . 165, 166

Dr. Theodore Fisher.

Seven Figures illustrating his paper on Peritoneal Sanguineous Cysts and their Relation to Cysts of the Pancreas . . . . 275—287

# NOTICE TO SUBSCRIBERS.

Terms of Subscription, including postage or delivery:

| | *s.* | *d.* |
|---|---|---|
| In Great Britain, nearly all the Colonies, and those Countries within the Postal Union . | 6 | 0 |
| To India . . . . | 7 | 0 |
| Terms to Non-subscribers . . . | 10 | 6 |

Subscriptions are due *immediately upon receipt of the volume.* Post-office orders should be drawn in favour of Dr. Hale White, made payable at the Vere Street Post Office, and addressed to 65, Harley Street, London, W.; they may with advantage be crossed "and Co."

A printed and numbered receipt will in all cases be sent to the Subscriber immediately on receipt of his remittance. If the Subscriber does not receive this within four days (except for foreign Subscriptions), he is requested to communicate at once with Mr. W. H. A. Jacobson, 66, Great Cumberland Place, W. In this way the Editors hope that all mistakes, of whatever kind, will be at once detected and investigated. Changes of address, or any other corrections in the list of Subscribers, should be forwarded to the Editors.

It is not, however, necessary to notify to the Editors each year the Subscriber's wish to continue on the list, as no name will be erased so long as the volumes are duly paid for, unless at the express desire of the Subscriber.

If any charge should be made for the delivery of this volume the Subscriber is requested to give information at once to the Editor.

# NOTICE.

Somewhat imperfect sets of the First and Second Series of the Reports can be had at very reduced prices on application to the Editors.

# LIST OF SUBSCRIBERS.

(Subscribers are requested to notify to the Editors any change of address.)

Abercrombie, J., M.D., 23, Upper Wimpole Street, W.
Aberdeen, Medico-Chirurgical Society, The Library, Medical Hall, 29, King Street, Aberdeen
Aberdeen University Library, Marischal College, Aberdeen
Adam, Mercer, M.D., The Churchyard, Boston, Lincolnshire
Adams, A. R., Royal Naval College, Greenwich
Adams, E. L,, Guy's Hospital
Adams, Matthew A., Ashford Road, Maidstone
Adeney, E. L., M.D., 3, Mount Sion, Tunbridge Wells
Aikin, C. E., 7, Clifton Place, Sussex Square, W.
Aikins, M. H., M.D., Burnhamthorpe, Ontario, Canada
Airy, H., M.D., Stoke House, Woodbridge, Suffolk
Alabone, M. A., Guy's Hospital
Alexander, H., Guy's Hospital
Alexander, K. B., Guy's Hospital
Alston, W. E., Guy's Hospital
Ambrose, A. C., Guy's Hospital
Anderson, C. T., Guy's Hospital
Anderson, G. E. C., M.D., Cape Town, Cape of Good Hope
Anderton, J. E., Thornfield, New Mills, Derbyshire
Andrew, E. G., Guy's Hospital
Andrews, Richard J., Homefield House, Heavitree, Exeter
Apthorpe, S. R., 1, Portland Terrace, Southampton.
Ashby, Alfred, M.B., Town Hall, Reading
Ashby, Henry, M.D., 31, St. John Street, Manchester
Ashwell, H. G., 2, Mansfield Road, Nottingham
Ashwin, R. H., Guy's Hospital
Askew, C. A. C., Guy's Hospital
Atkins, F. D., Chalk Pit House, Sutton, Surrey
Atkinson, T. R., Bainton House, Sherborne, Dorset
Audland, W. E., Brooklands, Wellingborough
Austen, H. H., M.D., Tandjong Poera, Langkat, Sumatra

Badcock, J. H., 140, Harley Street, W.
Bader, C., 10, Finsbury Circus, E.C.
Baines-Hooper, J. H., Guy's Hospital
Baker, A. E., 22, Grosvenor Street, W.
Balderston, R., Guy's Hospital
Baldwin, F. B. Judge, Cawdor House, Rotherham

Baldwin, H. R., M.D., New Brunswick, New Jersey, United States of America
Ball, J. A., M.B., Becket House, Wantage
Barker, H. J. M. D. S., Guy's Hospital
Barnes, J, E., J.P., Clare, Suffolk
Barrett, A. E., 24, Addison Terrace, Notting Hill, W.
Barrs, A. G., M.D., 22, Park Place, Leeds
Bartholomew, A. A., 22, London Street, Dockhead, S.E.
Bartlett, H., M.D., C.M., 150, Norwood Road, West Norwood
Barton, J. Kingston, 2, Courtfield Road, Gloucester Road, Queen's Gate, S.W.
Batchelor, F. C., 73, George Street, Dunedin, Otago, New Zealand
Batchelor, F. S , Guy's Hospital
Batchelor, H. C., Guy's Hospital
Beach, H. W., Guy's Hospital
Beadnell, C. M., Guy's Hospital
Beale, E. Clifford, M.B., 23, Upper Berkeley Street, Portman Square, W.
Bealey, Adam, M.D., Felsham Lodge, Hollington Park, St. Leonard's-on-Sea
Bearblock, P. E., Guy's Hospital
Beard, F., M.B., Brighton Road House, Croydon
Beatson, W. B., M.B., Vicarsgrange, Grange Road, Eastbourne
Beddard, A. P., B.A., Guy's Hospital
Beddard, F. E., M.A., F.R.S., Zoological Gardens, Regent's Park, N.W.
Bedford, G. H., Guy's Hospital
Beddoe, D. M., Guy's Hospital
Beeby, Walter T., M.D., Bromley, Kent
Beer, J., Guy's Hospital
Bell, H. T., Guy's Hospital
Bennett, C. V. S., J.P., Kensington House, Haverfordwest
Bennett, H., Builth, Breconshire
Berncastle, H. M., Guy's Hospital
Berry, H. P., M.B., 42, Watergate, Grantham
Berry, H. T., 66, Pembridge Villas, Bayswater
Berry, T. P., Guy's Hospital
Bettington, J. H., Guy's Hospital
Bevan, Richard, Cobb's Hall, Lydd, Kent
Beyts, W. G., Royal Victoria Hospital, Netley
Biddle, H. G., Beach House, Broadstairs
Biggs, J. J. E., Guy's Hospital
Biggs, T. S., Guy's Hospital
Bingham, S. (care of Dr. Hett, 100, Inverness Terrace, Bayswater, W.)
Bird, Tom, M.A., 59A, Brook Street, W.
Bird, W., Guy's Hospital
Birdwood, G. T., Royal Victoria Hospital, Netley
Birdwood, R. A., M.D., Hospital Ships, Long Reach, Dartford
Birkett, E. Lloyd, M.D., Westbourne Rectory, Emsworth, Hants
Birkett, John, 62, Green Street, Grosvenor Square, W.

Bisshopp, Francis R. B., M.A., M.D., B.C., Belvedere, Mount Pleasant, Tunbridge Wells
Bisshopp, J., Mount Ephraim, Tunbridge Wells
Blachford, J. V., 28, Avington Grove, Penge, S.E.
Black, G., M.B., 50, Cazenove Road, Stamford Hill, N.
Blackler, H. J., M.B., Guy's Hospital
Blaker, E. S., M.B., 24, Manor Road, Beckenham
Blaker, Nathaniel P., 29, Old Steyne, Brighton
Blaker, Thomas F. I., Wynnstay, Stanford Avenue, Preston Park, Brighton
Blaker, W. C., York House, Bognor
Blasson, G. H., Guy's Hospital
Blasson, Thomas, Billingborough, near Folkingham, Lincolnshire
Blatherwick, H., The Laurels, Dulwich, S.E.
Bligh, W., Guy's Hospital
Body, T. H., Guy's Hospital
Bolton, Alfred, J. P., 17, Wilson Patten Street, Warrington
Bond, C. S., B.A., Guy's Hospital
Booker, C. W., Guy's Hospital
Booth, E. H., M.D., 1, Cambridge Road, Hove, Brighton
Booth, Lionel, M.D., Sherburn House, Durham
Bossey, F., M.D., Mayfield, Redhill, Surrey
Boswell, J. I., M.D., Faversham, Kent
Bosworth, John Routledge, Sutton, Surrey
Bowden, G. H., Guy's Hospital
Bowen, O., 101, Heyworth Street, Liverpool
Bowes, J., 7, Marine Terrace, Herne Bay
Bradbury, J. A., 164, Wallgate, Wigan
Brailey, W. A., M.A., 11, Old Burlington Street, W.
Brawn, J. E. O., Guy's Hospital
Bredin, R., M.B., Valparaiso, Chili
Bremner, R. A., 47, Queen Street, Exeter
Brenton, W. H., 44, Cobourg Street, Plymouth
Brereton, F. S., Guy's Hospital
Brett, A. T., M.D., Watford House, Herts
Bright, John M., M.D., Alvaston, Park Hill, Forest Hill, S.E.
Brock, A. C., The Old House, Dorking
Brogden, R. W., M.B., B.S., 12, Lower Brook Street, Ipswich
Bromley, F. C., Guy's Hospital
Bromley, J. B., Castle Hedingham, Essex
Brookhouse, C. S., Guy's Hospital
Brookhouse, J. O., M.D., F.R.C.S., 1, East Circus Street, Nottingham
Brooks, B., Sonning, near Reading
Brown, Burton, M.D., 10, Queen Anne's Terrace, Cambridge
Brown, G. M., Guy's Hospital
Brown, R. T., Guy's Hospital
Brown, T., 236, Kennington Park Road, S.E.
Browne, George, 35, Montpelier Road, Brighton
Brussels, Académie Royale de Médecine de Belgique, Palais des Académies (per the Secretary)

Bryant, C. H., Guy's Hospital
Bryant, J. H., M.D., Guy's Hospital
Bryant, Thomas, M.Ch., 65, Grosvenor Street, W.
Buchanan, A. G., Guy's Hospital
Buchanan, Walter, Paddock House, Chatham, Kent
Bunting, James, Earlham, Torquay
Burdett, H. C., The Lodge, Porchester Square, W.
Burghard, F. F., M.S., 46, Weymouth Street, W.
Burrell, Edwin, M.D., 20, Endsleigh Street, Tavistock Square, W.C.
Burroughs, W. J., Guy's Hospital
Burrow, T., Guy's Hospital
Burton, C. G., Guy's Hospital
Burton, Herbert C., Lee Park Lodge, Lee, S.E.
Bush, F. P., Guy's Hospital
Bushell, S. Wootton, M.D. (care of R. Mathews, Esq., Bickley, Kent)
Busteed, J. H., Guy's Hospital
Butcher, J. O., 26, Harley Street, W.
Butler, A. G., Guy's Hospital
Butler, W. R., Guy's Hospital
Butler, William Harris, 15, Thomas Street, Woolwich
Butt, F. K., 9, Dalhousie Square, Calcutta
Button, Horace G., Blackall, Queensland

Cadel, N. P., Tiverton, N. Devon
Caldecott, C., M.B., The Grove, Jersey
Campbell, H. J., M.D., 25, King Henry's Road, N.W.
Cann, F. J. H., Guy's Hospital
Cann, Francis M., Sefton House, Dawlish, Devon
Cardiff Medical Society (per Dr. Paterson, Newport Road, Cardiff)
Cardin, H., Guy's Hospital
Carey, Francis, M.D., Villa Carey, Grange Road, Guernsey
Carling, W., B.A., M.B., B.C., Guy's Hospital
Carnelley, M., Woodley, Ruddington, near Nottingham
Carpenter, F. H., Guy's Hospital
Carpenter, S. H. M., Guy's Hospital
Carpmael, C. E., Guy's Hospital
Carr, T., Fallowfield, Manchester
Carter, A. H., Guy's Hospital
Cave, U. E., Guy's Hospital
Cawley, Thomas, M.D., North Terrace, Adelaide, South Australia
Cawston, E. A. S., Guy's Hospital
Cecil, J. E., Guy's Hospital
Chambers, T. R., Guy's Hospital
Chaning-Pearce, A., Guy's Hospital
Chapman, C. L. G., Guy's Hospital
Charles, H. E., 82, St. Helen's Road, Swansea
Cheese, James, 31, East Southernhay, Exeter
Chicken, Rupert C. (care of Mr. A. Bush, 43, Portland Road, Nottingham)

Chubb, W. L., Darenth House, Sandgate
Churchward, A., M.D., 206, Selhurst Road, South Norwood
Clague, J., Crofton, Castletown, Isle of Man
Clapham, Crochley, M.D., The Grange Asylum, near Rotherham, Yorks
Clapham, S. C., Guy's Hospital
Clark, A. W., 6, The Crescent, Wisbech, Cambridgeshire
Clark, R. F., Guy's Hospital
Clarke, A. E., Guy's Hospital
Clarke, A. V., Guy's Hospital
Clarke, Henry, H.M. Prison, Wakefield, Yorkshire
Clarke, W. T., Guy's Hospital
Clarke, W. E. J., Guy's Hospital
Clayton, E., Guy's Hospital
Clerment, Dr., 17, Rue St. Dominique, Paris (per Messrs. Hachette, 18, King William Street, W.C.)
Cleveland, A. J., Guy's Hospital
Cleveland, W. F., M.D., Stuart Villa, 199, Maida Vale, W.
Cloud, F. H. L., Guy's Hospital
Clowes, E. F., Guy's Hospital
Clowes, H. A., M.D., 2, Walterton Road, W.
Clowes, N. B., 18, Friar Street, Reading
Clunn, T. R. H., County Asylum, Prestwich, Manchester
Cock, F. W., M.D., 1, Porchester Houses, W.
Cock, H. E., Guy's Hospital
Cock, J., 17, Morton Crescent, Exmouth, Devon
Cock, W., 147, Queen's Road, Peckham, S.E.
Cogan, Lee F., 51, Sheep Street, Northampton
Colclough, W. F., B.A., Guy's Hospital
Cole, R. M., Northgate House, Gloucester
Coleman, E., Guy's Hospital
Coleman, F. J., Guy's Hospital
Coleman, J. G. B., Guy's Hospital
Coleman, J. J., Guy's Hospital
Collet, Aug. H., B.A., 1, Montague Place, Worthing, Sussex
Collett, A. J., Guy's Hospital
Collier, H. W., Guy's Hospital
Collington, F. A., College Villa, St. Helier's, Jersey
Collington, J. W., Kibworth, Leicestershire
Collins, H. A., The Chantry, Saxmundham
Collins, H. W., Wrington, near Bristol
Collins, V. E., Guy's Hospital
Collis, A. J., Guy's Hospital
Colson, Edward, Surgeon-Major, St. Helier's, Jersey
Compton, J. H., Guy's Hospital
Constant, F. C., Guy's Hospital
Cook, H., Guy's Hospital
Cooke, James Wood, Barnstaple, Devon
Cooper, H., B.A., M.B., B.Ch., Guy's Hospital
Couch, J. Q., 10, Chapel Street, Penzance
Couling, H., Grand Avenue Mansions, West Brighton

Coulson, J. E., Guy's Hospital
Counsell, H. E., Noname, East Liss, Hants
Court, Josiah, Staveley, Chesterfield
Cousins, J. S., Guy's Hospital
Coventry, C., Guy's Hospital
Covernton, C. E., Guy's Hospital
Creak, A. B., Guy's Hospital
Creasy, R., Windlesham, Bagshot, Surrey
Creed, C. P., Rathkeale House, Girdlers Road, West Kensington, W.
Cregeen, J. Nelson, Silverburn, 2A, Prince's Road, Liverpool
Cresswell, S. C., Guy's Hospital
Crew, E. J., Guy's Hospital
Crew, John, Manor House, Higham-Ferrers, Northamptonshire
Croft, John, 6, Mansfield Street, W.
Crompton, Dickinson W., 40, Harborne Road, Edgbaston, Birmingham
Croneen, A., Guy's Hospital
Crook, H. E., M.D., Carlton House, Dalby Square, Cliftonville, Margate
Crooke, G. F., M.D., 2, Edmund Street, Birmingham
Crosby, A. E. B., Guy's Hospital
Crosby, N. A., Guy's Hospital
Croydon Medical Book Club (per T. A. Richardson, Esq., care of Mr. Grattan, Tabard Book Store, Borough, S.E.)
Cruise, F. R., M.D., 93, Merrion Square West, Dublin
Cuff, H. E., M.D., The General Infirmary, Leeds
Cuff, R., M.B., 1, The Crescent, Scarborough
Cunningham, John, M.B., Campbeltown, Argyleshire
Currie, Andrew S., M.D., 81, Queen's Road, Finsbury Park, N.
Currie, O. J., M.B., Leslie, Westcombe Park, London, S.E.
Curtis, P. J., Guy's Hospital

Dakin, W. R., M.D., 57, Welbeck Street, W.
Daldy, A. M., M.B., B.S., Guy's Hospital
Dalton, B. N., M.D., Selhurst Road, South Norwood, S.E.
Dalton, C. B., Osmaston Road, Derby
Daly, N., Guy's Hospital
Daniel, W. Clement, M.D., Epsom, Surrey
Daniell, George Williamson, Blandford, Dorsetshire
Davies, A. G. C., Guy's Hospital
Davies, Andrew, M.D., Cefn Parc, Maindee, Newport, Mon.
Davies, Ebenezer, Brunswick House, Swansea
Davies, E. C., Guy's Hospital
Davies, F. W. S., Rainham, Kent
Davies, J., 91, New North Road, N.
Davies, T., Guy's Hospital
Davies, T. B. P., M.B., M.S., Guy's Hospital
Davies, W. R., Guy's Hospital
Davies-Colley, J. N. C., M.A., M.C., 36, Harley Street, W.
Davis, E. I., Guy's Hospital
Davison, T. S., Guy's Hospital

Davy, Henry, M.D., Southernhay House, Exeter
Dawson, W. J. O., Guy's Hospital
Day, F. N., Guy's Hospital
Day, T. M., Harlow, Essex
Deane, E., Greenham Villa, Caversham, Reading
De'Ath, Dr., Buckingham
Dell, J. F., 88, Dalston Lane, E.
Denman, R., Selborne, Poole Road, West Bournemouth
Devon and Exeter Hospital Library (per James Bankart, Esq., M.B., 19, Southernhay, Exeter)
Dix, John, 25, Albion Street, Hull
Dixon, G. F., Tyldesley, Manchester
Dodd, A. H., 14, Goldstone Villas, West Brighton
Dolman, A. J., Guy's Hospital
Donnelly, W. F. B., M.B., B.S., Guy's Hospital
Douglas, W. T. Parker, M.B., Newbury
Drew, H. W., 52, Dingwall Road, Croydon
Du Boulay, H. H., 2, Royal Terrace, Weymouth
Du Buisson, E. W., 85, Belvedere Road, Upper Norwood
Dudgeon, H. W., Guy's Hospital
Duffett, H. A., Guy's Hospital
Duncan, G. E., Guy's Hospital
Dunn, L. A., M.S., The College, Guy's Hospital, S.E.
Dupigny, J. E., Guy's Hospital
Duran, Carlos, and Nunez, Daniel, Costa Rica
Durbridge, H., Guy's Hospital
Durham, A. E., 82, Brook Street, W.
Durham, F., M.B., 82, Brook Street, W.
Durham, H. E., M.A., M.B., Guy's Hospital
Dutton, Edward G., Sherborne, 13, Spencer Terrace, Lipson Road, Plymouth
Dyson, J. N., Guy's Hospital

Eager, Reginald, M.D., Northwoods Asylum, Frampton Cotterell, near Bristol
East, W. N., Guy's Hospital
Eastes, F., M.D., 4, London Street, Folkestone
Eastes, George, M.B., 35, Gloucester Place, Hyde Park, W.
Eastes, T., M.D., 3, Shakespeare Terrace, Folkestone
Eccles, H. D., Guy's Hospital
Eddowes, A., Loughborough, Leicestershire
Edey, R. G., Guy's Hospital
Edwards, F. H., Guy's Hospital
Edwards, O., 34, Etnam Street, Leominster
Edwards, C. D., Guy's Hospital
Elder, George, M.D., C.M., 17, Regent Street, Nottingham
Elliott, C. C., M.B., B.S., Guy's Hospital
Elliott, E., The Gables, Bromsgrove, Worcestershire
Elliott-Blake, H., Kingsley, Bedford Square, Brighton
Elphinstone, R., Forest House, Silverstone, Towcester, Northamptonshire

Emmett, J. W., 19, Stodart Road, Anerley, S.E.
Emms, A. Wilson, J.P., Belgrave, Leicester
English, D.C., M.D., New Brunswick, New Jersey, United States of America
Evans, Alfred H., Sutton Coldfield, Warwickshire
Evans, C. R., Guy's Hospital
Evans, D. T., Three Counties Asylum, near Hitchin
Evans, Edward, Guy's Hospital
Evans, J. H., Broomfield, Crosby Road North, Waterloo, Liverpool
Evans, R. D., Guy's Hospital
Evans, T. H., Guy's Hospital
Evans, W. M., Hier, 80, Richmond Road, Cardiff
Evershed, Arthur, M.R.C.P.Lond., 29, Rosslyn Hill, Hampstead, N.W.
Evershed, A. R. F., Penzance, Cornwall
Evershed, Charles L., Arundel, Sussex
Every-Clayton, L. E. V., Guy's Hospital
Ewart, J. H., Eastney, Devonshire Place, Eastbourne
Ewen, H. W., Viewley, Redhill, Surrey
Eyre, J. W. H., Guy's Hospital

Fagge, C. H., Guy's Hospital
Fagge, R. H., Guy's Hospital
Fagge, T. H., M.D., 24, Clarges Street, W.
Faircloth, R., 3, Inverness Gardens, Kensington, W.
Falwasser, A. T., Guy's Hospital
Farnfield, W. W., Guy's Hospital
Farr, F. W., 167, Kennington Road, S.E.
Farr, George F., Slade House, 175, Kennington Road, S.E.
Fawcett, J., M.D., Guy's Hospital
Fawsitt, Thomas, 46, Union Street West, Oldham
Featherstone, G. W. B., Lynton Lodge, Cadishead, near Manchester
Fell, R., Guy's Hospital
Few, William, Ramsey, Huntingdon
Field, Ernest, M.D., C.M., 8, Belmont, Bath
Field, G. H., B.A., Guy's Hospital
Finch, A. H., Guy's Hospital
Fisher, Theo., M.D., 6, Oakfield Road, Clifton, Bristol
Fisher, W. H., Guy's Hospital
Fisk, E., Guy's Hospital
Fitz-Hugh, R. T., Guy's Hospital
Fleury, C. M., Guy's Hospital
Floyd, S. G., M.B., B.S., Guy's Hospital
Forman, E. Baxter, M.D., 11, Bramham Gardens, South Kensington, S.W.
Fortescue-Brickdale, J. M., Guy's Hospital
Fort, H. R. T., Guy's Hospital
Forty, D. H., Edbrook, Wotton-under-Edge, Gloucestershire
Foster, C. M., M.D., 1069, Yonge Street, Toronto, Canada
Foster, F. W., Thorpe-le-Soken, Colchester, Essex

Foster, J. J., Guy's Hospital
Foster, O. H., M.A., M.B., Hitchin, Hertfordshire
Foster, R. H., Knowle, Warwickshire
Fotherby, Henry I., M.D., Woodthorpe Cote, Reigate
Fowke, F. W., Byfield R.S.O., Northants
Fowke, W. C., Guy's Hospital
Fox, H. E. C., Guy's Hospital
Fox, H. W., Guy's Hospital
Fox, J. A., Tregea House, Morrab Road, Penzance
Francis, C. J., Guy's Hospital
Fraser, J. A., Western Lodge, Romford, Essex
Frazer, E. E., 4, John Street, Hampstead
Fripp, A. D., M.B., B.S., 65, Harley Street, W.
Frith, W. S., Guy's Hospital
Frost, F. T., Western Bank, Derby
Fry, A. C., B.A., Guy's Hospital
Fry, J. F., Shepton Mallet, Somersetshire
Fryer, E., Guy's Hospital
Fulham-Turner, H., Guy's Hospital
Fuller, Courtenay J., 37, Rectory Place, Woolwich
Fuller, Thomas, M.D., Longcrofts, New Shoreham, Sussex
Fuller, W. A., Guy's Hospital
Fulton, H., Guy's Hospital

Galabin, A. L., M.A., M.D., 49, Wimpole Street, W.
Galton, E. H., 128, Brixton Hill, S.W.
Galton, J. H., M.D., Chunam, 14, Sylvan Road, Norwood, S.E.
Gardner, J. T., 6, Hillsborough Terrace, Ilfracombe, Devon
Gardner, P. H., 1, Matlock Terrace, Torquay
Garland, J. O., Guy's Hospital
Garner, W. L., Guy's Hospital
Garrard, C. R. O., 30, Broad Street, Pendleton, Salford
Garrard, W. A., Chatham House, Rotherham, Yorkshire
Garrett, A. E., The Limes, Rickmansworth
Gathergood, B. W., Terrington St. John, Lynn, Norfolk
Gibson, J. H., 17, Grosvenor Road, Aldershot
Gibson, W., P. and O. Office, Leadenhall Street, E.C.
Gilford, H., F.R.C.S., Norwood House, Reading
Gillbard, R., Guy's Hospital
Gill, J. McD., M.D., Guy's Hospital
Girling, C. J., M.B. (care of Dr. A. E. Tayler, Bradbourne House, Acton, W.)
Glover, J. A., Guy's Hospital
Gocher, G., Guy's Hospital
Goddard, E. G., Guy's Hospital
Godfrey, T., Guy's Hospital
Godson, A. H., B.A., M.B., B.C., Guy's Hospital
Godson, F. A., B.A., Guy's Hospital
Godson, J. H., B.A., M.B., B.C., Guy's Hospital
Goldie, F. W., Guy's Hospital

Golding-Bird, C. H., M.B., 12, Queen Anne Street, Cavendish Square, W.
Golding-Bird, F., Guy's Hospital
Goodall, E. W., M.D., Homerton Fever Hospital, N.
Goodhart, J. F., M.D., 25, Weymouth Street, W.
Gorham, John, Bordyke Lodge, Tonbridge, Kent
Gosse, H. W., Eccleshall, Staffordshire
Gough, B. B., Guy's Hospital
Gowing, Benjamin C., Weirfield, Penistone, Yorkshire
Goyder, David, M.D., 88, Great Horton Road, Bradford, Yorkshire
Graham, G. H., Storrington, Pulborough, Sussex
Graham, S. G., Guy's Hospital
Granger, E. B., Grassington, near Skipton, Yorkshire
Graves, D. S., Guy's Hospital
Greaves, E. H., Guy's Hospital
Green, A., M.B., Burlington Street, Chesterfield, Derbyshire
Green, W. R., Guy's Hospital
Greenwood, E. Climson, 19, St. John's Wood Park, N.W.
Grellet, W. P., Guy's Hospital
Griffiths, D. R. T., Guy's Hospital
Gross, Charles, M.D., East Dulwich Grove, Champion Hill, S.E.
Grove, W. R., B.A., Guy's Hospital
Groves, C. E., F.R.S., Kennington Green, S.E.
Growse, J. L., Bildeston, Suffolk
Growse, W., Abbey Hill, Kenilworth
Gruggen, W., 11, Montpelier Road, Ealing, W.
Guy's Hospital Library (Two Copies)
Guy's Hospital Museum (care of Curator)

Habershon, S. H., M.D., 70, Brook Street, Grosvenor Square, W.
Haden, W. H., M.D., 66, Harley Street, W.
Hall, E. S., Guy's Hospital
Hall, F. W., M.D., M.S., Esher Woollahra, Sydney, N.S.W.
Hall, James Griffith, J.P., Prospect Place, Swansea
Halliwell, T., 7, Argyle Villas, Brockley Road, Forest Hill, S.E.
Ham, B. B., Guy's Hospital
Hancock, E. D., Guy's Hospital
Hancock, W. I., Guy's Hospital
Handley, W. S., Guy's Hospital
Hardenberg, E. F. H., Guy's Hospital
Harding, C. H., Guy's Hospital
Hardy, F. H., Guy's Hospital
Harnett, C. J., Guy's Hospital
Harper, C. J., Church End, Finchley, N.
Harris, E. B., Tottenham Lane, Hornsey
Harris, J. W., Cranbrook, Kent
Harris, P. Traer, 112, Harley Street, W.
Harris, R., M.B., 18, Duke Street, Southport
Harris, W. J., B.A., M.B., B.C., Guy's Hospital
Harrison, A. J., M.B., Failand Lodge, Guthrie Road, Clifton
Harrison, S., Guy's Hospital

Harsant, J. G., M.D., The Hive, Exeter Road, Bournemouth
Harsant, W. H., 16, Pembroke Road, Clifton, Bristol
Hartree, J. P., M.B., Leigh, Tunbridge
Hayes, T. E. D., M.D., High Street, Reigate
Hayward, John W., Whitstable, Kent
Hazell, F., M.B., Guy's Hospital
Heath, F. H. R., Guy's Hospital
Heddy, William Jackson, 46, Redcliffe Gardens, S.W.
Henderson, E. E., Guy's Hospital
Henry, P. F., Guy's Hospital
Henson, W. W., Guy's Hospital
Hetley, Henry, M.D., Beaufort House, Church Road, Norwood, S.E.
Hewetson, H., Guy's Hospital
Hibbard, C. E., Guy's Hospital
Hickman, H. V., M.B., Wanstead
Hicks, H. T., Guy's Hospital
Hicks, John Braxton, M.D., F.R.S., 34, George Street, Hanover Square, W.
Hicks, R., 126, High Street, Ramsgate
Higgens, C., 38, Brook Street, W.
Hills, A. Phillips, Carlton House, Prince of Wales Road, Batterse Park, S.W.
Hills, William Charles, M.D., Thorpe St. Andrew, near Norwich
Hills, W. E., Guy's Hospital
Hind, Wheelton, M.D., Stoke-on-Trent
Hindle, F. T., Hill Croft, Askerne, Doncaster
Hinds, H. A., Guy's Hospital
Hinton, H. T., Guy's Hospital
Hitchins, F. C., Guy's Hospital
Hobson, J. M., M.D., Glendalough, Morland Road, Croydon
Hodgson, C. R., Guy's Hospital
Hodson, Frederic, Hornsea, Hull
Holman, C., M.D., J.P., Reigate
Holman, H. J., Lydfords, East Hoathley, Sussex
Holman, R. C., M.D., Midhurst, Sussex
Holman, T. E., Guy's Hospital
Holmes, T., M.B., Forton Villa, near Garstang
Holmested, T., 12, Freeland Road, Ealing, W.
Hood, Donald W. Charles, M.D., 43, Green Street, Park Lane, W.
Hopkins, C. L., B.A., Guy's Hospital
Hopkins, F. G., B.Sc., Guy's Hospital
Hopson, M. F., Grove House, Rosslyn Hill, Hampstead
Hore, Surg.-Capt., A.M.S., Malta
Horrocks, P., M.D., 26, St. Thomas's Street, S.E.
Horsley, H., 42, London Road, Croydon
Hort, E. C., B.A., Guy's Hospital
Horton, J. H., Guy's Hospital
Horton, W. A., Guy's Hospital
Hovenden, A. C., Guy's Hospital
Hovenden, G. S., Guy's Hospital

Howard, J. A., Guy's Hospital
Howe, A. F. A.,Guy's Hospital
Howe, J. D., Bank House, Croston, near Preston
Howell, J. B., 14, High Street, Wandsworth, S.W.
Howell, T. A. I., Cleeve House, West Hill, Wandsworth, S.W.
Howitt, H. G., Guy's Hospital
Howse, H. G., M.S., 59, Brook Street, W.
Hubbard, Fred. E., The Lawn, Diss, Norfolk
Hubbard, Thos. Wells, Rock House, Boughton Monchelsea, Maidstone, Kent
Huckle, A. H. H., Guy's Hospital
Hughes, Robert Harry, M.A., M.B , 12, Lockyer Street, Plymouth
Hughes, S., Guy's Hospital
Hughes, W. L., Guy's Hospital
Hugill, Geo. F., Arundel Lodge, Balham, S.W.
Hull, A. J., Guy's Hospital
Humphreys, F. R., 27, Fellows Road, N.W.
Humphreys, Fredk. Wm., F.R.C.S., 24, Sinclair Gardens, West Kensington, W.
Hunter, P. D., Guy's Hospital
Hurst, L. H., Guy's Hospital
Hutson, E., Guy's Hospital

Ibotson, E. C. B., Guy's Hospital
Ince, John, M.D., Montague House, Swanley, Kent.
Inñeld, S., Guy's Hospital
Ingle, Arnold C., B.A., M.B., B.C., 21, Regent Street, Cambridge
Instone, B., Guy's Hospital
Isaac, B., Guy's Hospital
Izard, H. E., Guy's Hospital

Jackson, Arthur, 17, Wilkinson Street, Sheffield
Jackson, P. J., 216, Great Dover Street, S.E.
Jackson, T., M.D., 6, Scotch Street, Whitehaven
Jackson, T. L., B.A., Guy's Hospital
Jackson, T. S., Guy's Hospital
Jacobson, T. B., Sleaford, Lincolnshire
Jacobson, W. H. A., M.A., M.Ch., 66, Great Cumberland Place, Hyde Park, W.
Jalland, W. H., St. Leonard's House, York
James, W. C., M.D., 11, Marloes Road, Cromwell Road, Kensington, W.
Jaynes, V. A., 157, Jamaica Road, S.E.
Jenkins, C. C., Guy's Hospital
Jennings, Oscar, M.D, 33, Rue Marbœuf, Avenue des Champs Elysées, Paris
Jensen, E. T., Guy's Hospital
Jewell, J. W. F., Guy's Hospital
Jiminez, G., Guy's Hospital
John, H. W., Ammanford, R.S.O., S. Wales
John, Wm., 19, Avonmore Road, West Kensington, W.

John, T., Guy's Hospital
Johnson, W., Guy's Hospital
Johnson, W. J., Guy's Hospital
Jones, A. H., M.D., 45, Sheep Street, Northampton
Jones, B., Leigh, Lancashire
Jones, G. H. West, Southgate, Eckington, Rotherham
Jones, G. M., Rawdon House, Alton, Hants
Jones, G. W., Guy's Hospital
Jones, H. J., 167, Lillie Road, S.W.
Jones, J. Edwards, M.D., Bryn-y-ffynon, Dolgelley, North Wales
Jones, J. H., Guy's Hospital
Jones, Robert, 22, Great George Square, Liverpool
Jones, R. O., Guy's Hospital
Jones, T., M.B., 89, Mosely Street, Manchester
Jones, W. Makeig, Wath-upon-Dearne, Rotherham
Joslen, H., Arcadia Pen, Porus, P.O., Jamaica
Joyce, J. H., B.A., M.B., B.C., Guy's Hospital

Kay, Dr. N. E. M., 23, Brunswick Street, Halifax, Nova Scotia
Kay, R., Guy's Hospital
Keall, C. A. H., Guy's Hospital
Kelbe, W. E., Idutywa, Transkei, Cape Colony
Kellock, W. B., Stamford Hill, Stoke Newington, N.
Kelsey, A., Redhill, Surrey
Kelso Dispensary, Roxburghshire (per Dr. Thomas Hamilton)
Kemp, G. L., Worksop, Notts
Kendall, G., Battle, Sussex
Kendall, Walter B., Greenheys, King's Wear, Devon
Kent, Thomas J., 89, Piccadilly, W.
Ker, Hugh Richard, Devonshire Cottage, Balham Hill, S.W.
Kerby, R. J., Speenhamland, Newbury
Key, B. W. M. A., Guy's Hospital
Kidd, W. A., M.D., B.S., 12, Montpelier Row, Blackheath, S.E.
King, C., 23, Highbury Road, N.
King, T. P., B.A., Guy's Hospital
Kingsford, Edward, Sunbury, Middlesex
Kingsford, E. C., Carntyne, Brondesbury, N.W.
Kinsey-Morgan, A., Bournemouth
Kinsey-Morgan, A., Guy's Hospital
Kitching, C. M., M.D., Cape Town, Cape of Good Hope
Knaggs, R. L., M.A., M.D., M.C., 37, Park Square, Leeds
Knight, E. V., Guy's Hospital
Knight, F. C. R. M., Guy's Hospital
Knight, J. T., Carlton, near Nottingham
Knipe, W. M., Melbourne, Derbyshire
Knott, Charles, Elm Grove, Southsea
Koëttlitz, R., Butterknowle, Darlington

Lacy, A. G., The Cottage, Sunninghill, Ascot, Berks
Lamb, Hugh, "Heatherlie," Ashton-on-Ribble, Lancashire

Lamb, William Henry, M.B., 23, Palace Court, W.
Lambert, F. E. L., Guy's Hospital
Lancaster, H. F., M.D., 154, Westbourne Terrace, W.
Lancereaux, E., M.D., 44, Rue de la Bienfaisance, Paris
Landon, E. E. B., Guy's Hospital
Lane, H., 11, The Circus, Bath
Lane, W. Arbuthnot, M.S., 8, St. Thomas's Street, S.E.
Lansdown, F. P., Samber House, 19, White Ladies' Road, Clifton Bristol
Larkin, F. G., Grove Park, Lee, Kent
Larking, A. E., Chesham, Bucks
Latimer, H. A., 4, Belvoir Place, Swansea
Lavers, N., Guy's Hospital
Lavrenius, A. N., Guy's Hospital
Lawrence, G., Guy's Hospital
Leathes, J. B., B.A., M.B., B.Ch., Guy's Hospital
Lawton, W. C., Guy's Hospital
Layman, S. G., Guy's Hospital
Leader, H., Guy's Hospital
Lee, C. G., 73, Rodney Street, Liverpool
Lee, F. W., Guy's Hospital
Leeds School of Medicine Library (per the Secretary of Yorkshire College, Leeds)
Leigh, W. W., Treharris, South Wales
Lever, F., M.B., B.S., B.Sc., Montpellier Lodge, Harrogate
Levick, G. K., Guy's Hospital
Lewis, Ivor A., Cymmer, Porth, Glamorganshire
Lewis, L. H., Guy's Hospital
Lichtenberg, C. L., Guy's Hospital
Lidderdale, F. J., Guy's Hospital
Lipscomb, E. H., M.B., St. Albans, Herts
Lister, T. D., M.B., Guy's Hospital
Littlewood, J. O., Sutton-in-Ashfield, Nottinghamshire
Lloyd, M., M.D., Vale Villa, Llanarthney, R.S.O.
Lockwood, J. P., Faringdon, Berkshire
Long, D. S., M.B., B.C., Guy's Hospital
Longhurst, E. A., Guy's Hospital
Longhurst, S. H., Guy's Hospital
Longmore, Sir T., C.B., The Paddock, Woolstone, Southampton
Lord, P., M.B., Royal Naval Hospital, Haslar
Loud, F., Guy's Hospital
Love, A. E. B., Richmond Villa, Bournemouth
Loveday, W.D., Stoney Stratford, Bucks
Lowe, P. R., B.A., Guy's Hospital
Lower, C. A., Guy's Hospital
Lower, N. Y., Guy's Hospital
Lucas, C. R., Guy's Hospital
Lucas, H., Huntingdon
Lucas, R. Clement, B.S., 18, Finsbury Square, E.C.

Luce, R. H., B.A., M.B., B.C., Guy's Hospital
Lund, E., 22, St. John Street, Manchester
Luscombe, T. B., Cromer House, Teddington
Lush, Wm. George Vawdrey, M.D., 12, Frederick Place, Weymouth
McCarthy, J., McC., St. George's, Wellington, Salo
McIlroy, W., Guy's Hospital
MacIlwaine, S. W., South Petherton, Somerset
Mackern, George, M.D., Calle Tucuman 571, Buenos Ayres
Mackern, J., M.D., St. German's Lodge, Blackheath, S.E.
Maconchy, John K., M.B., Infirmary House, Downpatrick
Maggs, W. A., 16, Hanover Square, W.
Mahomed, A. G., Astolat, Bournemouth
Major, H. G. T., B.A., Guy's Hospital
Mallam, G. B., 102, Iffley Road, Oxford
Mallam, W. P., 169, Uxbridge Road, Shepherd's Bush, W.
Manby, Alan R, M.D., East Rudham, Norfolk
Manby, E. P., M.D., South Dispensary, Liverpool
Manchester Royal Infirmary (per The Secretary)
Manley, J. H. H., M.A., M.B., 20, New Street, West Bromwich
Mann, C. J. H., Guy's Hospital
Manning, G. E., Guy's Hospital
Manning, T. D., Guy's Hospital
Mansbridge, J., 112, Harley Street, W.
Mansell, E. R., 46, Wellington Square, Hastings
Manser, F., The Priory, Tunbridge Wells
March, E. G., Guy's Hospital
Marriott, E. B., Guy's Hospital
Marriott, Hyde, B.Sc., M.B., The Limes, Hall Street, Stockport
Marriott, H. B., Walton, Ipswich
Marriott, O., Guy's Hospital
Marsh, H. R., Guy's Hospital
Marshall, Edward, Mitcham, Surrey
Marshall, E. W., Church House, Mitcham, Surrey
Marshall, J., 143, Grange Road, S.E.
Marshall, W. L. W., New North Road, Huddersfield
Martin, Albert, M.D., Ingestre Street, Wellington, New Zealand
Martin, F., M.D., Holmhurst, Southend Road, Beckenham, Kent
Martin, F. J. H., Guy's Hospital
Martin, James P., Lorne Villa, Box, Wilts
Mason, S. H., Guy's Hospital
Matcham, Alfred, 116, St. George's Road, Southwark, S.E.
Mathews, Robert, Bickley, Kent
Mathison, A. J., Guy's Hospital
Matthews, A. K., Guy's Hospital
Maturin, A. E., Guy's Hospital
Maxwell, E. J., Guy's Hospital
May, E., M.D., Fairholme, Hanwell, Middlesex
Mayston, R. W., Guy's Hospital
Meares, A. L. D. (care of Miles Bros., 79, Gracechurch Street, E.C.)
Medwin, A. G., M.D., 34, Bruton Street, Berkeley Square, W.

Mesquita, S. B. de, M.B., B.S., 113, Petherton Road, Highbury New Park, N.
Metcalfe, G. H., Clare, Suffolk
Meyer, C. H. L., M.D., Grant Medical College, Bombay
Meyrick-Jones, H., Guy's Hospital
Mickley, George, M.B., M.C., St. Luke's Hospital, Old Street, E.C.
Milbank-Smith, H. J., Guy's Hospital
Miller, A. A., Guy's Hospital
Miller, H. R., Guy's Hospital
Milligan, R. A., 50, Sheep Street, Northampton
Mills, T. I., Guy's Hospital
Milton, W. T., Guy's Hospital
Milward, J., M.D., Cardiff
Mitchener, M. N., Guy's Hospital
Moffatt, C. E., Guy's Hospital
Moffatt, H. A., Guy's Hospital
Moon, R. H., 160, Norwood Road, West Norwood, Surrey
Moon, R. O., Guy's Hospital
Moore, J., Guy's Hospital
Moore, P. W., Guy's Hospital
Moore, W. H., 18, Church Street, Kidderminster
Morgan, E., Guy's Hospital
Morice, C. G., Greymouth, New Zealand
Morison, Joshua W., Hamilton Terrace, Pembroke, South Wales
Morris, Henry, M.A., M.B., 8, Cavendish Square, W.
Morrison, J. T. J., M.B., 27, Newhall Street, Birmingham
Morse, T. H., 10, Upper Surrey Street, Norwich
Mossop, C. H., Guy's Hospital
Mothersole, R. D., M.D., M.S., 23, Head Street, Colchester
Mottram, G. N., Guy's Hospital
Mugford, S. A., Manningtree, Essex
Mühlenkamp, F., Guy's Hospital
Mules, J. H., Guy's Hospital
Mumford, W. G., Guy's Hospital
Muncaster, J. H., B.A., M.B., Guy's Hospital
Munden, C., Ilminster, Somerset
Munro, H. A., B.A., Guy's Hospital
Muriel, Charles Evans, 36, St. Giles's Street, Norwich
Muriel, George John, 109, Scotch Street, Whitehaven
Murphy, S. F., 41, Queen Anne Street, W.
Murray, C. H., Guy's Hospital
Muspratt, C. D., M.D., 19, Lansdowne Road, East Cliff, Bournemouth

Nason, J. J., M.B., Church House, Stratford-on-Avon
Nevins, J. E., M.B., 3, Abercromby Square, Liverpool
Nevill, P. H., Guy's Hospital
Newland-Pedley, F., 32, Devonshire Place, W.
Newnham, W. H. C., M.B., 1, Leicester Place, Clifton, Bristol
Newton, J. L., M.D., 90, Newtown Road, Sydney, New South Wales
Nicholls, F. J., Guy's Hospital

Nicholson, C. R., Guy's Hospital
Nicholson, T. M., Guy's Hospital
Nisbett, Robert Innes, The Eagles, Overcliffe, Gravesend
Norburn, A. E., M.B., 26, Cazenove Road, Stamford Hill, N.
Norman, A., Guy's Hospital
Northampton General Infirmary Library (per the House Surgeon)
Nourse, A. W., Guy's Hospital
Nunneley, John, M.B., 22, Park Place, Leeds

Odling, William, M.A., Ph.D., M.B., F.R.S., 15, Norham Gardens, Oxford
Ogle, C. J., 1, Cavendish Place, W.
O'Grady, E. S., M.B., C.M., 33, Merrion Square North, Dublin
Oldham, C. J., 38, Brunswick Square, Brighton
Oldham, Henry, M.D., 4, Cavendish Place, W.
Oldman, C. E., M.D., The Grange, Bletchingley, Surrey
O'Meara, E. J., Guy's Hospital
Onraët, H. Bernard, Guy's Hospital
Oram, R. R. W., Cremyll, Wandsworth Common, S.W.
Ormond, A. W., Guy's Hospital
Otway, C. C., 13, Kennington Park Road, S.E.
Owen, S. Walshe, 10, Shepherd's Bush Road, W.
Owsley, G. C., Guy's Hospital
Oxley, W., 1, South Terrace, Rotherham, Yorkshire

Packer, H. D., Guy's Hospital
Padley, George, Northampton Villas, Swansea
Paget, P., General Hospital, Leicester
Pakes, W. C., Guy's Hospital
Palmer, Clement, Burton-under-Needwood, Burton-on-Trent
Palmer, F. J. M., M.B., "Christowell," Riggindale Road, Streatham, S.W.
Palmer, H. D., Stourbank, Nayland, Colchester
Palmer, J. Irwin, 47, Queen Anne Street, W.
Pantin, C. S., M.B., B.S., Guy's Hospital
Paramore, Richard, M.D., 2, Gordon Square, W.C.
Pare, J. W., M.D., Guy's Hospital
Parfitt, J. B., Guy's Hospital
Park, W. C. C., Guy's Hospital
Parkin, A., M.D., M.S., 5, Albion Street, Hull
Parkinson, C. H. Watts, Wimborne, Dorset
Parry, R., M.B., Castle Street, Carnarvon
Parsons, J. E. H., B.A., Guy's Hospital
Partridge, A. A. H., M.A., Guy's Hospital
Passmore, A. B., Guy's Hospital
Paul, Frank T., 38, Rodney Street, Liverpool
Paul, L. A., Guy's Hospital
Pavy, F. W., M.D., F.R.S., 35, Grosvenor Street, W.
Pawle, M. G., Guy's Hospital

Payne, Arthur J., M.D., Surgeon-Major, Bengal Medical Service (per Messrs. Lewis & Co., Gower Street)
Peake, W. H., Guy's Hospital
Pearse, E. Sainthill, 3, Albion Street, Brierley Hill, Staffordshire
Pearson, G. B., Guy's Hospital
Peatson, J. C., Guy's Hospital
Pedley, J. K., Guy's Hospital
Pedley, S. E., 10, The Terrace, Camberwell
Pegge, Charles, Vernon House Asylum, Briton Ferry, Glamorganshire
Peile, H. D., Guy's Hospital
Pelly, Surgeon-General S. M., C.B., Woodstock House, Burnt Ash Hill, Lee, S.E.
Pendlebury, J. P., Knowles House, Ormskirk
Pendred, B. F., Guy's Hospital
Pendred, V., Guy's Hospital
Penfold, F. W. H., Rainham, Sittingbourne
Pennington, S. A. B. C. C., Guy's Hospital
Penny, E., M.D., Hermitage, Marlborough
Percival, G. H., M.B., 66, Abington Street, Northampton
Perkins, Geo. Steele, M.B., 26, Weymouth Street, W.
Perkins, H. B., Llysmeddyg, Newport, R.S.O., Pembroke
Perks, A., Guy's Hospital
Perks, R. H., M.D., Adelaide, South Australia
Pern, H., Guy's Hospital
Perry, E. C., M.A., M.D., Guy's Hospital
Peters, E. A., B.A., Guy's Hospital
Philipps, A. E., Guy's Hospital
Phillipps, W. A., M.D., 13, John Street, Berkeley Square, W.
Phillips, H. A., M.D., 39, The Terrace, Gravesend
Phillips, R. E. G., Guy's Hospital
Philps, F. G. M., Guy's Hospital
Picnot, E., Guy's Hospital
Pike, N. H., Guy's Hospital
Pigeon, H. W., M.A., M.C., F.R.C.S., 6, Albion Street, Hull
Pilcher, W. H., Guy's Hospital
Pilcher, W. J., High Street, Boston, Lincolnshire
Pilkington, F. S., 172, Norton Street, Leichhardt, N.S.W.
Pilkington, F. W., County Asylum, Littlemore, Oxon
Pilkington, George, 5, St. Peter's Place, Fleetwood, Lancashire
Pillin, H. L., 33, George Street, Hanover Square, W.
Pinching, Charles J. W., 76, New Road, Gravesend
Pitt, G. Newton, M.A., M.D., 24, St. Thomas's Street, S.E.
Pitt, W. C., Guy's Hospital
Plimmer, H. G., Wunderbau, Norwood, S.E.
Plomley, John Fred., M.D., 9, West Boro', Maidstone
Plummer, W. E., Guy's Hospital
Poland, John, 4, St. Thomas's Street, S.E.
Pollard, C., M.D., Fulham Road, S.W.
Pollock, J. R. R., Cheveley House, Newmarket
Pomeroy, E. H., Guy's Hospital

Poole, T. B., Guy's Hospital
Poolman, A. E., B.A., Castle Street, Abergavenny, Monmouth
Pope, Edward, Tring, Herts
Portsmouth Medical Library (care of F. Lord, Esq., 16, Landport Terrace, Southsea)
Powell, A. W., Guy's Hospital
Prall, S. E., Guy's Hospital
Prendergast, J. M., M.D., 26, Boulevard Malesherbes, Paris
Price, A. E., M.D., 8, High Street, Bromley, Kent
Price-Jones, C., 7, Claremont Road, Surbiton
Pritchard, R. J., Guy's Hospital
Proctor-Sims, F. R., Guy's Hospital
Purves, W. Laidlaw, M.D., 20, Stratford Place, Oxford Street, W.
Puzey, Chauncy, 71, Rodney Street, Liverpool
Pye-Smith, P. H., B.A., M.D., F.R.S., 48, Brook Street, W.
Pye-Smith, R. J., 350, Glossop Road, Sheffield
Pywell, W. H., 244, Westminster Bridge Road, S.E.

Radley, R. W. J., Guy's Hospital
Rake, A. T., M.B., B.S., Guy's Hospital
Rake, B. N., M.D., The Leper Asylum, Trinidad
Rake, H. V., "St. Ives," Fordingbridge, Salisbury
Ramskill, Josiah, 29, Meadow Lane, Leeds
Rand, John, Glenmore, Underhill Road, Lordship Lane, S.E.
Randall, P. N., H.M.S. Warspite, Pacific Station
Randell, R. M. H., M.D., Copers Cope Road, Beckenham
Ransford, T. D., F.R.C.S., 6, Queen's Square, Bath
Ray, Edward Reynolds, Dulwich Village, S.E.
Rayson, H. K., Guy's Hospital
Reed, J. C. G., Guy's Hospital
Reeves, A., Guy's Hospital
Reeves, J. K., B.A., 1, Craster Road, Brixton Hill, S.W.
Regnart, H. C., Guy's Hospital
Reid, E., Guy's Hospital
Reinold, A. W., M.A., F.R.S., 84, Vanbrugh Park, Blackheath, S.E.
Rendell, W., Maiden Newton, Dorset
Reynolds, B. G., Guy's Hospital
Reynolds, L. G., Guy's Hospital
Reynolds, W. P., M.D., 128, Stamford Hill, N.
Richards, G. O., Onslow House, Richmond Green
Richardson, E. H., M.D., 31, 1-2 Peachtree Street, Atlanta, U.S.A.
Richardson, H., 114, Highbury New Park, N.
Richardson, T. A. (care of Mr. Grattan, Tabard Book Store, Borough, S.E.)
Ricketts, T. F., M.D., Hospital Ships, Long Reach, Dartford, Kent
Rigby, James A., M.D., 25, Winckley Square, Preston
Rigg, V. J. J., Guy's Hospital
Rix, B., 2, Mount Ephraim Road, Tunbridge Wells
Roberts, Sir Alfred, 125, Macquarie Street, Sydney, New South Wales
Roberts, Astley C., Badlesmere, Eastbourne

Roberts, Bransby, M.D., Badlesmere House, Eastbourne, Sussex
Roberts, C. Gordon, M.A., M.B., Halstead, Essex
Roberts, H. J., Penygroes, Carnarvon
Roberts, H. W., 166, Lewisham High Road, S.E.
Roberts, J. Lloyd, M.D., 110, Prince's Road, Liverpool
Roberts, R. J., M.B., The Beeches, Lowestoft
Robertson, J., Guy's Hospital
Robinson, F. W., 6, New North Road, Huddersfield
Robinson, W. E., B.A., Guy's Hospital
Robson, E. Sheddon, B.A., The Bailey, Durham
Rogers, E. Coulton, Fulbourn Asylum, Cambridge
Rogers, W. G., M.S., M.B., Eastern Fever Hospital, Homerton, E.
Romano, F. W. R., Pelotas, Rio Grand do Sul, Brazil
Rooke, Thomas Morley, M.D., 7, Bays Hill Villas, Cheltenham
Roose, Robson, LL.D., M.D., 45, Hill Street, Berkeley Square, W.
Roots, W. H., Kingston-on-Thames
Roper, A., M.D., Colby, Lewisham Hill, S.E.
Roper, A. L., M.A., Guy's Hospital
Roper, E., Highfield House, Shepperton, Middlesex
Roper, G., M.D., Oulton Lodge, Aylsham, Norfolk
Ross, Richard, M.D., 7, Wellington Place, Belfast
Routh, C. F., M.D., Uppingham, Rutland
Rouw, R. W., 7, Wimpole Street, W.
Rowell, G., 7, Harley Street, W.
Rowell, H. E., East Rudham, Norfolk
Rowland, F. W., Guy's Hospital
Rowland, L. T. A., Guy's Hospital
Rowland, R. J., Guy's Hospital
Rowland, R. S., Guy's Hospital
Rowland, W. J., B.A., Guy's Hospital
Rowlands, R. P., Guy's Hospital
Russell, J. W., M.B., General Hospital, Birmingham
Ruzzak, S. A., Guy's Hospital
Ryle, A. B., Guy's Hospital
Ryle, R. J., M.B., Hadley, Barnet

Sadler, H. G., 3, St. George's Fields, Canterbury
Saffrey, F. G., Guy's Hospital
St. Mary's Hospital, Manchester (care of the Librarian)
Salter, C. E., M.B., B.S., Guy's Hospital
Salzmann, Frederick William, 97A, Montpellier Road, Brighton
Sandoe, J. W., M.D., B.S., Broad Clyst, near Exeter
Sangster, Charles, 148, Lambeth Road, S.E.
Sargent, G. P., M.D., 5, The Mart, Station Road, Brixton, S.W.
Savage, G. H., M.D., 3, Henrietta Street, Cavendish Square, W.
Schofield, G., Durham House, Bournemouth
Scott, A., M.D., Fulham Union Infirmary, Hammersmith, W.
Scott, A. L., Westbourne Villa, Ealing Dean, W.
Scott, B., Hartington, Bournemouth
Scott, E. N., Guy's Hospital

Scott, Francis, East Dene, Grove Road, Bournemouth
Scott, R. J. H., 28, Circus, Bath
Scott, W. J., M.D., B.S., 18, Castellain Road, Maida Hill, W.
Scowby, E. T., Guy's Hospital
Sells, C. B., Guy's Hospital
Sells, C. J., White Hall, Guildford
Sells, Hubert T., Northfleet, Kent
Serjeant, R., Guy's Hospital
Shadwell, St. C. B., Lyndhurst, Orford Road, Walthamstow
Sharp, A. J., M.B., B.S., Guy's Hospital
Sharp, H. C., B.A., M.B., B.C., Guy's Hospital
Sharp, John Adolphus, Charnwood House, Osmaston Road, Derby
Shaw, C. Knox, 19, Upper Wimpole Street, W.
Shaw, F. H., 33, Warrior Square, St. Leonards
Shaw, H. T., Guy's Hospital
Shaw, L. E., M.D., 10, St. Thomas's Street, S.E.
Sheen, A. W., M.B., B.S., Guy's Hospital
Sheffield Medico-Chirurgical Society (Dr. W. S. Porter, 269, Glossop Road, Sheffield)
Sheldon, T. Steele, M.B., Parkside Asylum, Macclesfield
Shells, W. F. M., 5, Davey Street, Hobart, Tasmania
Shelswell, O. B., 8, Glebe Villas, Mitcham
Shillingford, H., The Limes, Peckham Rye, S.E.
Shillingford, F., Chesterfield House, Esplanade, Weymouth
Shillingford, H. B., 57, Rye Lane, Peckham, S.E.
Shillitoe, A., B.A., Guy's Hospital
Shipman, George Wm., Grantham, Lincolnshire
Shorland, E. T., Guy's Hospital
Shute, G. S., M.D., 2, Granby Place, Northfleet, Kent
Sigler, Geo. A., M.D., Liberty, Union County, Indiana, United States of America
Silk, J. F. W., M.D., 29, Weymouth Street, W.
Simon, R. M., B.A., M.D., 47, Newhall Street, Birmingham
Skinner, David S., M.D., Westbury House, Harrow Road, Harlesden, N.W.
Slater, L., B.A., Guy's Hospital
Smallpeice, W. D., 42, Queen Anne's Gate, S.W.
Smith, G. B., Farnah, Bromley Road, Lee, S.E.
Smith, H. Austen, M.B. (care of H. S. King and Co., 65, Cornhill, E.C.)
Smith, H. E. M., Guy's Hospital
Smith, H. J. M., Guy's Hospital
Smith, H. V., Guy's Hospital
Smith, J. Snowden, Tavistock, Devon
Smith, James William, 13, Hall Gate, Doncaster
Smith, W. A. L., B.A., Guy's Hospital
Smith, W. H. M., Guy's Hospital
Soper, William, 307, Clapham Road, S.W.
South London Medical Reading Society (per Bramley Taylor, M.D., 145, Denmark Hill, S.E.)
Spicer, A. H., Guy's Hospital

Spon, H. J., Guy's Hospital
Spriggs, E. Ivens, Guy's Hospital
Spurgin, Herbert B., 82, Abington Street, Northampton
Spurgin, Thomas, Manor House, Ongar, Essex
Spurgin, W. H., Maryport, Cumberland
Spurrell, C., Eastern Fever Hospital, Homerton, E.
Stabb-Johnson, E. W., Guy's Hospital
Stamford, R. B., Guy's Hospital
Stamper, James F., M.D., Pembroke Dock, South Wales
Stanton, T., Guy's Hospital
Starling, E. A., M.B., 65, Calverley Road, Tunbridge Wells
Starling, E. H., M.D., 14, Grosvenor Road, Westminster, S.W.
Starling, H. J., Guy's Hospital
Statham, C. B., Guy's Hospital
Statter, H. B., Snapethorpe Hall, Wakefield
Steinhaeuser, J. R., Guy's Hospital
Stephen, L. H. Y., Guy's Hospital
Stephens, L. E. W., Emsworth, Hants
Stephens, Thomas Palmer, Emsworth, Hants
Stevens, G. J. B., 1, Newington Green, N.
Stevens, R. Ingram, Hoddesdon, Herts
Stevens, T. G., M.D., Evelina Hospital, Southwark Bridge Road, S.E.
Stevenson, T., M.D., Sandhurst Lodge, 45, Gresham Road, Brixton, S.W.
Steward, F. J., Guy's Hospital
Stewart, H. M., B.A., Guy's Hospital
Still, G. F., M.A., M.B., B.C., Guy's Hospital
Stocker, John Sherwood, M.D., 2, Montagu Square, W.
Stoke Newington, Clapton, and Hackney Medical Book Society (per F. Wallace, Esq., Foulden Lodge, Upper Clapton)
Stoner, H. B., Guy's Hospital
Stothard, W. J., 70, Palatine Road, Didsbury, Manchester
Strasburg University Library
Stuart, E. O., Albion House, Newtown, Stockport
Stuart, S. O., Medical Staff Mess, Aldershot
Stuart, W. L., Guy's Hospital
Sturge, H. H., Belmont Lodge, New Eltham, Kent
Sturges-Jones, W. E., 39A, Plough Road, Battersea
Suffolk Medical Book Society, The Ancient House, Ipswich
Sutton, C. R. A., B.A., M.B., B.C., Guy's Hospital
Sutton, Frederick, Willingham-by-Stow, Gainsboro', Lincolnshire
Swayne, J. G., M.D., 74, Pembroke Road, Clifton, Bristol
Swayne, W. C., M.B., 3, Leicester Villas, St. Paul's Road, Clifton, Bristol
Sweetlove, J. W., Guy's Hospital
Symonds, C. J., M.D., M.S., 26, Weymouth Street, W.

Targett, J. H., M.S., 6, St. Thomas's Street, S.E.
Taylor, A. S., M.B., Elmleigh, Surbiton Hill
Taylor, D., 163, Clapham Road, S.W.

Taylor, E. C., Guy's Hospital
Taylor, Frederick, M.D., 20, Wimpole Street, W.
Taylor, H. Owen, M.D., Pelham Crescent Park, Nottingham
Taylor, J. W., Guy's Hospital
Taylor, W. Bramley, 145, Denmark Hill, S.E.
Telling, W. H. M., Guy's Hospital
Temple, P. G., Guy's Hospital
Terry, E. B., Woodchurch, near Ashford
Terry, J. G., Wittersham, Kent
Thomas, A., M.B., B.S., Guy's Hospital
Thomas, A. M., Guy's Hospital
Thomas, Jabez, Ty Cerrig, Swansea
Thomas, J. D., Guy's Hospital
Thomas, T. M., Guy's Hospital
Thorne, J. M., Burntwood Lodge, Upper Tooting, S.W.
Thorneley, M. H., Guy's Hospital
Thornton, R., Guy's Hospital
Thorp, C. G., 29, Southwark Bridge Road, S.E.
Thorpe, G. E., Knight, 77, Gell Street, Sheffield
Thorpe, W. G., M.D., 20, Larkhall Rise, Clapham, S.W.
Ticehurst, A. R., Winstowe, St. Leonards
Ticehurst, C. S., The Nook, Petersfield
Tilling, W. H. M., Guy's Hospital
Tilley, Samuel, F.R.C.S., 1, Down Street, Piccadilly, W.
Todd, D. B., Guy's Hospital
Todd, G., Sydenham, Torquay
Tongue, E. J., Guy's Hospital
Toulmin, W., 102, Elm Park Gardens, S.W.
Townsend, T. S., 68, Queen's Gate, South Kensington, S.W.
Tresidder, M. E., Guy's Hospital
Tresidder, P. E., Guy's Hospital
Treusch, J., Guy's Hospital
Trevor, E. T., 3, St. James's Street, Buckingham Gate, S.W.
Trick, W. H., Guy's Hospital
Tubby, A. H., M.S., 39, Finsbury Circus, E.C.
Tuchmann, M., M.D., 113, Fellows Road, N.W.
Tulk-Hart, T. J. A., Guy's Hospital
Turner, A. Scott, Guy's Hospital
Turner, F. Charlewood, M.A., M.D., 15, Finsbury Square, E.C.
Turner, F. Meadows, M.B., Guy's Hospital
Turner, G., M.B., Rosslyn, Broxbourne, Herts
Turner, H. Gunton, Holmwood, Bournemouth
Turner, J. Sidney, 81, Anerley Road, Anerley, S.E.
Tyrrell, A., Guy's Hospital
Tyson, W. J., M.D., 10, Langhorne Gardens, Folkestone

Uhthoff, J. C., M.D., 9, Brunswick Place, Hove, Brighton
Underhill, W. Lees, The Limes, Alcester Road, Moseley, Birmingham
Underwood, J. C., Guy's Hospital
University College Library, London, Gower Street, W.C.

Van Someren, E. H., Guy's Hospital
Veasey, Henry, Aspley-Guise, Woburn, Bedfordshire
Vellacott, P. N., Guy's Hospital

Wacher, F., Monastery House, Canterbury
Wacher, S., Dane Town, Canterbury
Waddy, H. E., Rhossili, Brunswick Road, Gloucester
Wagner, A. L., M.D., South Bend, Indiana, St. Joseph Co., U.S.A.
Wainewright, R. S., M.D., 39, Lee Terrace, Blackheath, S.E.
Wakefield, C. F., Guy's Hospital
Wales, H. J. L., Guy's Hospital
Wales, T. Garneys, Downham Market, Norfolk
Walker, F. E., Guy's Hospital
Walker, T. M., Guy's Hospital
Wallace, R. U., M.B., Cravenhurst, 148, Stamford Hill, N.
Waller, W. A. E., Rugby
Walling, H. W. B., Guy's Hospital
Wallis, F. H., Guy's Hospital
Wallis, H., Guy's Hospital
Wallis, W., jun., Groombridge, Tunbridge Wells
Walters, J. B., Guy's Hospital
Walters, J. H., Guy's Hospital
Ward, J. L. W., Merthyr Tydfil
Ward, P. H., Guy's Hospital
Warner, P., Rydal, Woodford Green, Essex
Washbourn, J. W., M.D., 30, Trinity Street, S.E.
Wason, R. L., 19, West Street, Buckingham
Watson, C. E. S., Guy's Hospital
Watson, W., Rochester
Watt, J. G., Guy's Hospital
Waymark, W. E., Guy's Hospital
Weaver, F. P., M.D., Cedar Lawn, Hampstead Heath, N.W.
Webb, G. B., Guy's Hospital
Weber, Hermann, M.D., 10, Grosvenor Street, W.
Weekes, C. P., Guy's Hospital
Weir, Patrick A., M.A., M.B., C.M., Surgeon-Major, The Residency, Nepal, India
Welford, F., B.A., M.B., Guy's Hospital
Wernet, A. J., Guy's Hospital
Weston, E. F., The Green Hall, Stafford
Wheatcroft, E. W. (care of Dr. Grellit, Hitchin)
Wheeler, D., Chelmsford
Whicher, S., Guy's Hospital
White, W. Hale, M.D., 65, Harley Street, W.
Whitworth, G., Guy's Hospital
Wilkin, J. F., M.D., M.C., The Warren, Beckenham Park, Kent
Wilkins, J. C. V., Guy's Hospital
Wilkinson, J. C., 1, Elwick Road, Ashford, Kent
Wilks, H. L. E., The Infirmary, Salisbury

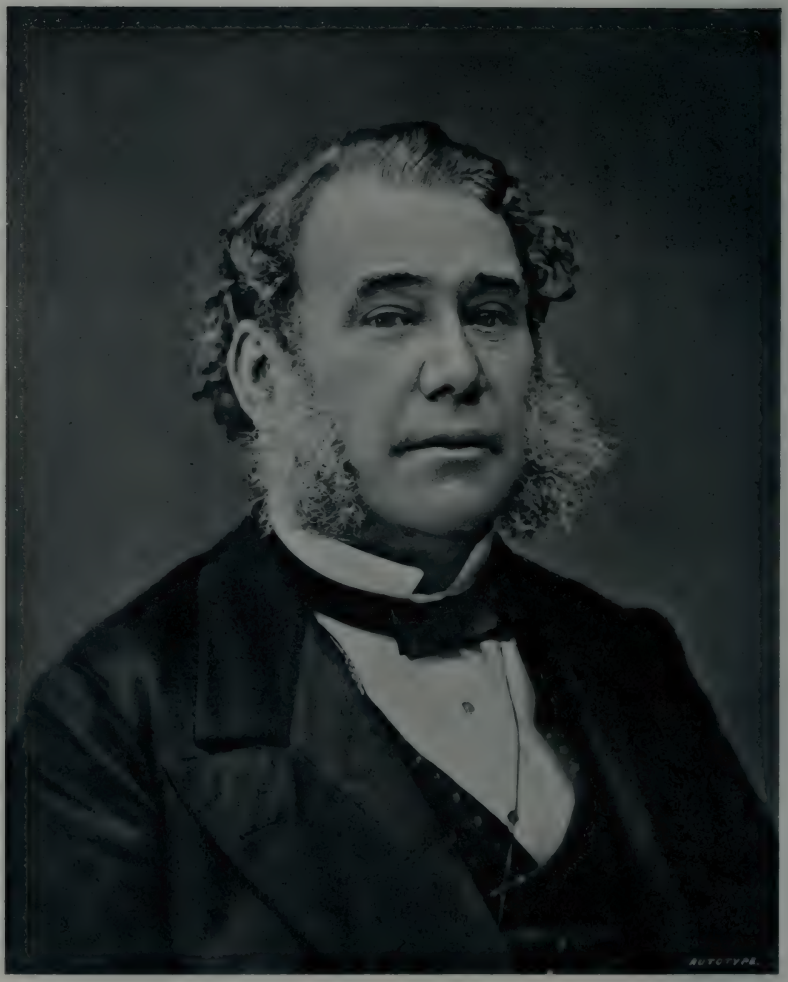
Wilks, Samuel, M.D., LL.D., F.R.S., 72, Grosvenor Street, Grosvenor Square, W.
Willan, G. T., Melton Mowbray, Leicestershire
Willett, E. Sparshall, M.D., Wyke House, Syon Hill, Isleworth
Williams, A. H., 54, London Road, St. Leonards-on-Sea
Williams, R. E., M.B., 21, The Common, Woolwich
Williams, W. R., M.D., Linden House, Bertle Road, Leamington
Williamson, N., M.D., New Brunswick, New Jersey, United States of America
Willis, G. N., Guy's Hospital
Wilmot, P. McK., Guy's Hospital
Wilson, W., M.B., C.M., 184, Goldhawk Road, Shepherd's Bush, W.
Wilson-Smith, T., M.D., Kidbrooke Lodge, Oldfield Park, Bath
Wilton, J., M.D., Wrecclesham, Farnham, Surrey
Winckworth, C. E., Shefford, Beds.
Wingate, W. W., B.A., Jesus College, Cambridge
Winslow, W., Crake Hall, Wimbledon Common, S.W.
Winter, C., Guy's Hospital
Winter, L. B., Guy's Hospital
Wohlmann, A. S., M.D., Royal Mineral Water Hospital, Bath
Wood, F. S., Westbourne House, Sheffield
Wood, P. M., Esher, Holden Street, Ashfield, Sydney, N.S.W.
Wood, W. R., Guy's Hospital
Woodhouse, A. E. C., 1, Hanover Square, W.
Wordley, A. W., 8, Great Suffolk Street, S.E.
Worthington, H. E., Guy's Hospital
Worthington, S., M.D., Rectory Place, Woolwich
Worts, E., Colchester, Essex
Worts, L., Guy's Hospital
Wright, G. A., M.B., 8A, St. John Street, Manchester
Wright, H. H., 2, Ospringe Road, St. John's College Park, N.W.
Wyatt-Smith, F., M.B., B.C. (care of R. J. Neild, Esq., 6, Finsbury Circus, E.C.

York Medical Society (care of Richard Turner, Esq., 1, Low Ousegate)

## In Exchange.

The St. Bartholomew's Hospital Reports.
The St. Thomas's Hospital Reports.
The Westminster Hospital Reports.
The Edinburgh Medical Journal (care of Messrs. Oliver and Boyd, Tweddale Court, Edinburgh).
The Dublin Journal of Medical Sciences (care of Messrs. Fannen and Co., Grafton Street, Dublin).
The Birmingham Medical Review (care of Dr. Saundby, 83A, Edmund Street, Birmingham).
The Bristol Medico-Chirurgical Journal (care of L. M. Griffiths, Esq., 9, Gordon Road, Clifton, Bristol).
The Liverpool Medico-Chirurgical Journal (care of Dr. James Barr, 72, Rodney Street, Liverpool).
The Medical Chronicle (care of Dr. Williamson, 294, Oxford Road, Manchester).
Sheffield Medical Journal (care of Simeon Snell, Esq., 17, Eyre Street, Sheffield).
The Medical Magazine (care of Messrs. Southwood Smith and Co., 4, King Street, Cheapside, E.C.).
The Pharmaceutical Journal.
The Transactions of the Hunterian Society.
The Transactions of the Obstetrical Society of London.
The Practitioner (care of Macmillan and Co.).
Library of Surgeon-General's Office, U.S. Army, Washington, D.C. (per Mr. B. F. Stevens, U.S. Government Despatch Agency, 4, Trafalgar Square, London, W.C.)
The American Journal of the Medical Sciences (care of Messrs. Lee Bros. and Co., Philadelphia, U.S.A.).
The Journal of Nervous and Mental Diseases (care of Dr. C. H. Brown, 25, West 45th Street, New York).
Transactions of the Odontological Society (care of the Hon. Sec., Odontological Society, 40, Leicester Square, W.C.).
Transactions of the College of Physicians, Philadelphia, U.S.A.
The Albany Medical Annals (per Messrs. Burdick and Taylor, 481, Broadway, Albany), care of Smithsonian Institute, Washington, D.C., U.S.A. [* Parcel to be labelled "free of duty."]
The Medical News (care of Lea Brothers & Co., 706 & 708, Sansom Street, Philadelphia, U.S.A.).
John Hopkins Museum, Baltimore, U.S.A.
Southern Medical Record (care of Dr. D. H. Howell, Atalanta, Ga., U.S.A.).
Revue des Sciences Médicales en France, et à l'Étranger (care of M. le Dr. Hayem, 17, Rue du Sommerard, Paris).

Le Progrès Médical (care of Dr. Bourneville, Rue des Écoles 6, Paris).
Revue de Médecine (108, Boulevard Saint Germain, Paris).
Annals of the Pasteur Institute (Le Bibliothècaire, Institut Pasteur, Rue Dulot, Paris).
Mémoires de la Société de Médecine et de Chirurgie de Bordeaux (care of Dr. Demons, Hôpital St. André, Bordeaux).
Archives d'Électricité médicale (care of M. J. Bergonié, 6 bis Rue du Temple, Bordeaux).
Le Bulletin de la Société d'Anatomie et de Physiologie de Bordeaux (care of M. le Dr. X. Arnozan, 27 bis, Pavé des Chartons, Bordeaux).
Les Archives de Physiologie Normale et Pathologique (care of Mons. Brown-Séquard, 120, Boulevard Saint Germain, Paris).
Verhandlungen der Berliner medicinischen Gesellschaft (care of Herr B. Fränkel, Bibliothek der Berliner medicinischen Gesellschaft Ziegelstrasse, 10, Berlin. N.).
Centralblatt für Chirurgie (care of Messrs. Breitkopf und Härtel, Leipzig)
Centralblatt für klinische Medicin (care of Messrs. Breitkopf und Härtel, Leipzig).
Upsala Läkareförenings Förhandlingar (per Prof. Hedenius, Bibliothèque de la Société des Médecins, Upsal, Suède).
Il Progresso Medico, Largo Pignasecca 7, Naples.
Il Policlinico, Turin (care of Professor Dr. Carlo Forlanini, Turin).


AUTOTYPE.

# In Memoriam.

## JOHN HILTON, F.R.S.

As no obituary notice of a Surgeon to whom Guy's owes very much has ever appeared in these pages, we have availed ourselves of an opportunity which the death of Mr. Cock—so long associated with Mr. Hilton—has given of making an attempt, though very late in the day, of supplying this omission in our Reports.

As the writer only knew Mr. Hilton during the last years of his life, he would take this opportunity of thanking, cordially, those from whom he received much valuable information as to earlier years.

Much of whatever good there may be in this article must be placed to the credit of the following:—Dr. Pavy, Dr. Pye-Smith, Dr. Goodhart, Dr. Savage, Dr. Constantine Holman, Dr. T. Alpheus Buck, Mr. Birkett, Mr. Durham, Mr. Davies-Colley, and Mr. Couling.

John Hilton was born September 22nd, 1807, on historic ground, at Sible Hedingham in Essex. Two small villages, Sible Hedingham and Castle Hedingham, lie, a little north of Halstead, on the Colne Valley branch of the Great Eastern Railway. The two villages[1] are adjoining, but separated by the Colne. Castle Hedingham, on the east bank, was the chief stronghold of the De Veres, Earls of

[1] In Sible Hedingham churchyard several stones bear the name of Hilton. Anyone visiting the Hedinghams may, by striking west, across the "Suffolk Way," or the great road between the Roman stations at London and Caister, visit the church of Hempstead, and the grave of Harvey.

Oxford, whose crest of the blue boar (Verres), badge of the mullet, and motto (*Vero nihil verius*) are familiar to all who have seen anything of the Churches and Halls of the Eastern Counties. Here Hilton's eyes must have been early familiar with the great Norman tower, the fine bridge over the ditch, and the old trees on the side of the rising steep which is covered by the remains of the castle. In Sible Hedingham, on the other side of the river, lived Sir John Hawkwood, one of the unscrupulous free-lances of the fourteenth century, who, after beginning life as a tailor, was knighted by Edward III, and afterwards became a leader of Condottieri in the wars of Italy. In return for his services Florence erected a monument to his memory, which may still be seen in her cathedral. Figures of hawks carved in different parts of the English church show that the church was rebuilt either by Sir John or one of his name.

Hilton was educated at the Grammar School at Chelmsford, and at Boulogne-sur-Mer. Some years later, when, as Demonstrator of Anatomy, he revisited the latter place for a holiday, an event occurred which shows what changes have taken place in the last seventy years. While travelling on the top of a diligence Hilton found himself seated next a man in whom, now sleek and prosperous, and dressed in most respectable broadcloth, he recognised one who had retired a few years before from the business of body-snatcher. Conversation promptly followed, and in reply to a question as to what had brought him into France, the body-snatcher replied that he had come over to fetch back one of his daughters who was at school "over there." Hilton, who knew the place, said, "But they only learn a sort of *patois* there." To this the reply was, "Oh no, sir, my daughters learn French and the *patois* too."

Hilton was uncertain whether this worthy was one of the brothers Harnetts or the brothers Crouch. The doings of this quartette of scoundrels are chronicled in the volumes of Bransby Cooper's life of his uncle, in the memorials of J. F. South, and in a diary which, though little known, is perhaps the most remarkable of any that this world has ever contained, 'Diary of a London Resurrectionist, 1812–13,' Royal College of Surgeons' Library. How one of the

brothers Crouch was called down from London "in consultation" by Liston (then settled in Edinburgh) to aid him in obtaining the body of a country lad, who, after dying of hydrocephalus, had been buried in a graveyard on the shores of the Firth of Forth; how, for weeks, the friends had succeeded in baffling the body-snatchers; how, when the excitement of the contest between the watchers and the besieging force of body-snatchers had begun to wane, Liston and Ben Crouch came down on the scene, and in thirty minutes of twilight, just before the watchers appeared on the ground, succeeded in carrying off the body, which found a final resting-place in the Museum at Edinburgh—all this will be found told in Dr. Lonsdale's 'Life of Robert Knox,' p. 66. Mr. South states—let us hope incorrectly, and unduly biassed by the rapid success with which Guy's had ousted her elder sister—that the Crouches were the sons "of the carpenter of Guy's Hospital."

Hilton's education seems to have been inadequate and incomplete, but the having to pick up things as he could probably early sharpened his natural shrewdness and acumen. At all events, Hilton's life in this respect is a proof that when a man honestly wins success without the help that a good education, money, or powerful friends can give, there must be something in that man to stamp him as above the common—much in him to honour. And we may go a step further here: it is certain that Hilton's rough outside, his want of personal attractiveness, his reserved, hard manner, and his aversion to society save that of his friends, all pointed to the fact that the early and most impressionable years of his life had been overclouded, and that he had not come out of that time unharmed.

We have no information of Hilton's student days, which must have been about the years 1824 to 1828. One story he left behind which shows how men and manners have changed. The separation between Guy's and St. Thomas's took place in 1825, but a yearly dinner was held at which the staff and their friends met—a meeting still continued under the auspices of the United Hospital Club. In one of his later student years Hilton had an invitation. Men drank hard in those days, and Hilton used to tell how, as

chair after chair became vacated, either by the guests leaving or finding repose under, the table,[1] he moved up until he reached a seat of honour at one side of the chairman, the elder Benjamin Travers.[2] It would be interesting to know what passed between the pair. Hilton could not remember how Travers took the intrusion; he only recollected that after getting back to his lodgings in Maze Pond and going to bed he woke up in the middle of the night, desirous of getting some water "to cool the tip of his tongue." His disgust may be imagined when he stepped on to a soaking carpet, and on looking for the cause at his finding that a fellow-student, who lodged in the same house, had come into his bedroom to get some water, and that he was sitting in one corner of the room pouring water out of Hilton's bedroom ewer on to the bottom of a tumbler which he held upside down, while he anathematised his thirst and his inability to quench it.

Hilton was appointed Demonstrator of Anatomy in 1828, the second time this appointment had been made at Guy's. Mr. Cock had been the first, having been appointed in 1825, when the two hospitals were separated. As Hilton was without interest, it is clear that he must have already made his mark to have this appointment given him when he was only twenty-one.[3] The time at which the appointment came to him is of importance, as it was at this time that the Guy's

[1] This dinner must have been about 1828; it recalls a story related by Dean Ramsay in his 'Reminiscences of Scottish Life and Character,' as coming from Mackenzie, the author of 'The Man of Feeling,' who lived from 1745 to 1831. It shows that the Scotch were as canny in their prevention of apoplexy or suffocation as they were ultra-hospitable in pressing guests to drink. "He had been involved in a regular drinking party. He was keeping as free from the regular excesses as he was able, and as he marked companions around him falling victims to the power of drink, he himself dropped off under the table amongst the slain, as a measure of precaution, and lying there, his attention was drawn to a small pair of hands working at his throat. On asking what it was, a voice replied, 'Sir, I'm the lad that's to louse the neckcloths.'"

[2] One of the earliest of the scientific, or, as they have been called, physiological surgeons, author of an 'Inquiry into the Process of Nature in repairing Injuries of the Intestines, illustrating the Treatment of Penetrating Wounds and Strangulated Hernia,' 1812, and 'Inquiries concerning Constitutional Irritation and the Pathology of the Nervous System,' 1826 and 1835.

[3] I think it may be taken as a fact that Hilton was the first of those appointed in our School or on the Surgical Staff without having been first apprenticed to one of the Surgeons.

Medical School was in its infancy, having been opened in 1825, the final rupture between us and St. Thomas's taking place in 1836. In 1832 the Anatomical Act came into force and replaced the business of the body-snatchers, and during these years there was a third and popular school in the Borough, Grainger's, competing with Guy's and St. Thomas's. The above must have combined to make these exciting times. How differences sprang up between Guy's and St. Thomas's about 1824, how a medical school was first started, independently, at Guy's in 1825, and how the tie between the united Borough hospitals was finally broken in 1836,—all this is well told in the life of Sir A. Cooper by his nephew Bransby (vol. ii), in the memorials of Mr. South, and by Dr. Wilks in his 'Biographical History of Guy's Hospital.' It was a happy storm which cleared the air and started our school on its own lines, with Mr. Cock as the first demonstrator in 1825, and Mr. Hilton associated with him three years later. Mr. Hilton was at this time living in Maze Pond, and if, as sometimes happened, a body was brought after the hours when the dissecting room was closed, it was placed in the cellar of the house that Hilton was occupying.

Hilton was Demonstrator for fourteen years, and was reported to be the best anatomist in London.[1] It was these years which laid the foundation on which his name will rest. The dissecting room of that day now forms part of our Museum. The Demonstrator of those times must have been more important in some ways than at the present. There was none of that profusion of well-illustrated books at the disposal of all, at moderate prices. There was but one examination at the end of the three years, at which Anatomy had to be taken up with other subjects. Anatomy in 1828 occupied an infinitely more important position in the early training of a medical student than it does now. An attempt had only just been made, and very imperfectly, to separate it from Physiology. This blending of the two subjects showed itself in the basis of very sound if very simple Physiology which runs through all Hilton's teaching. It was natural under the circumstances that it should first appear in his earlier anatomical teaching; it was constantly

[1] Dr. Pye-Smith.

coming out, later on, in his surgical work, both in the wards and in his lectures, unpublished as well as published. It is not going too far to say that it is in this sound blending of Anatomy and Physiology, coupled with the natural powers of the man, that the chief merit of Hilton's teaching lies. It is on this that his imperishable claim to be remembered as a great scientific or physiological surgeon rests, strange as it may appear to many who only remember Hilton's outer man to hear the term scientific applied to him, and still more so, to those who have been trained in the physiology of 1893, to hear Hilton called physiological. We shall return to this matter later. We have alluded to it here because some of the chief colouring and the chief characteristics of Hilton's teaching date to the years with which we are dealing, and the relation which Anatomy occupied to Physiology in the year 1828. Hilton, in his earlier years in the dissecting room certainly, worked at Anatomy as no one at Guy's has ever worked either before his day or since; and the chief result is to be seen in the models with which his name and those of Towne the artist and Harrison the treasurer should always be associated. Hilton is said to have lived about the place all day, taking his lunch in a room which should correspond to the present prosector's room. It was this devotion to his subject which won him the agnomen of "Anatomical John."[1]

He must have been thoroughly at home with his subject, having it at his fingers' ends; a teacher in whom, having confidence in himself and his knowledge of the subject, students had entire confidence in all he said, as one who was teaching them that kind of Anatomy which would be useful to them hereafter in the practice of their profession. The work of Demonstrator and of students alike must have been easier in that day. There was much less competition between hospitals and their schools. There were far fewer lectures and demonstrations, and thus teachers and students alike had more time and more energy to give to that which lay before them; each man worked more on his own lines, and less by rules laid down by Universities and Colleges. Students in qualifying for the examination of that day had to rely more

[1] Dr. Pavy.

on their own exertion, and to obtain their knowledge more by exercising their own efforts, instead of relying upon their teachers to give them just what was wanted in neatly made sandwiches or as spoon-meat which required no mental mastition. If when the time of examination came a man suffered shipwreck in those days, it was on the long-known coast of Little Knowledge, and not on the more recent quicksands of Over-Cram.

The event which should fix clearly in the minds of Guy's men 1828 as the year of Hilton's appointment as Demonstrator of Anatomy was the arrival in London about this time of one who was destined to do work with Hilton which, while proving of infinite service to generation after generation of our students, has, in the lapse of years, been too much separated from the names of the three men to whom we owe it. It was in 1826 that young Towne came to London from Royston with his model of a human skeleton, called upon, and was kindly received by Mr., afterwards Sir, A. Cooper, who was so pleased with the accuracy that he gave him a letter of introduction to Mr. Harrison, the Treasurer. This man, to whom Guy's owes so much, who succeeded his father as Treasurer at the early age of twenty-six, who founded a separate medical school at Guy's; who, because he was in some ways despotic and arbitrary, was called "King Harrison," but to whose zeal and interest in the working of every detail in our school its earliest successes were largely due;—this man, who, by sheer power of judging merit, appointed three of those whose names will confer most abiding lustre on Guy's in the future—Addison, Hilton, and Gull—to Harrison's memory students of the present day should turn in reverence, respect, and gratitude for this also, that he, by his power of judging men, and by his foresight and knowledge of what would benefit our school—a knowledge in which he must have been immensely in advance of his day—gave us also those models in the possession of which our Museum has been without a rival, and the envy of other schools. What arrangement passed between Mr. Harrison—who had then been Treasurer for twenty-nine years, and who was not to retire until 1848, after a reign of fifty-one years—and Joseph Towne (who, then only seventeen years old, was to work for our

hospital for fifty-three years) will never be known. It was a day of an absolute despotism, which held its sway, on the whole most usefully, regardless of the modern checks which the institution of auditors has given. Mr. Bryant, in his notice of Mr. Towne in our Reports for 1882, writes, "All Towne bargained for was a room to himself, on which he could turn a key when he liked, and the promise of every kind of help from Mr. Harrison, should help be required, in the unknown work he had undertaken to originate." Later on an arrangement was made by which his salary was paid partly by the Hospital, partly by the School—an arrangement which lasted up to 1853, after which date until his death in 1879 the school paid it all. Amongst his earlier models are his best, the anatomical ones which represent the first attempt made in this country to represent the different regions of the human body by wax, and amongst the first of these to be undertaken was the series of thirty sections to illustrate all the parts of the brain. Then followed the models made from dissections, and, as in the case of those just mentioned, by the hand of John Hilton. Before this time, owing to the difficulty of obtaining subjects for dissection, wax models had been introduced into England from Florence and Paris, some of which can still be seen in Museums in London and at the older Universities. Some familiarity with these foreign models is necessary in order to estimate rightly the value of those with which the wisdom of Harrison and the labour of Hilton and Towne have enriched our school. In the models of foreign artists it is comparatively little that is attempted to be shown, the different structures are very poorly defined, the skill of the anatomist to show and the power of the artist to represent are alike at fault; while the perishable nature of the materials used, and the tendency of the tints to become faded and blurred become increased as time goes on. On the other hand, it is difficult to know which to praise most in the handiwork of Towne—their life-like accuracy, their pains-taking determination to represent details faithfully, the truthful sharpness of outline by which the different structures represented are separated one from the other, and, perhaps, above all,

the abiding permanence of the colour and materials employed.

No one will ever know now how these marvellous models were made. Not only did Towne's skill and proficiency die with him, not only was his power—the peculiarity of a true artist of fixing in wax or marble what he had once seen in life or death—lost when he departed, but, unfortunately for our profession, he left behind him no account of the improvements he introduced into the materials and colours which he used, and more particularly how he perfected the permanence of our wax models. This was largely due to his solitary habits and love of working alone. One of the conditions which he made with Mr. Harrison was, as we have seen, that he should have a room which he could keep locked. This room was in earlier days one that became later the old Materia Medica museum. Later on it was one attached to the Clinical buildings. In either case all that men saw was Towne coming or going, and an old red-capped assistant that looked like an Italian. The way in which Hilton did his share of the work was to devote an hour or two every day to dissecting with the minutest care a very small part—merely one or two inches[1]—of some region on which he was engaged. He then left, and Towne with like accuracy and faithfulness copied those dissections. When it is remembered that this rule was carried on daily all the year round, in days before the present modes of injection and preservation had come into vogue, it speaks volumes for the energy and perseverance of the fellow-labourers.

During the later years in which Hilton was Demonstrator of Anatomy he was also associated with Mr. King in making the post-mortem examinations, and in giving demonstrations in Morbid Anatomy. This part of the field at Guy's has been so long occupied by a series of distinguished physicians, that it is interesting to remember, now, how Hilton's tenure of the post influenced his life's work. It is seen in the care with which he unravelled the course of the cerebro-spinal fluid, investigated the causation of hydrocephalus in children, and traced it in many cases to closure of the cerebro-spinal aperture in the roof of the fourth ventricle. The same

[1] Mr. Birkett.

familiarity with necropsies and the desire to search out the truth which they alone give is shown repeatedly in Hilton's 'Rest and Pain,' in his account of injuries to the head, his explanation of sudden death which sometimes follows on disease of the upper cervical vertebræ; and so it is throughout the book up to the very end, to the very last case with which it closes. It will be remembered that this was a patient whom Hilton saw, once, for the effects of a severe sprain to one ankle which was followed by fatal suppuration between the shaft and the lower epiphysis of the tibia. Hilton's account runs:—"I did not again see this patient alive. Five or six days afterwards he died with what was thought to be typhoid fever. By chance, on the day before the funeral, I heard of his death. I immediately wrote to his relations and obtained permission to make an examination of the leg. I went to the house early on the following morning; the hearse was at the door, so I had only time to unscrew the coffin and examine the leg. The periosteum was separated from the inner, anterior, and posterior part of the lower portion of the tibia by a considerable collection of pus, which was confined in its position by the periosteum." Hilton must have been over fifty-five when he thus posted up to Islington, intent on clearing up a case. Dr. Wilks refers to his meeting Hilton in his younger days at a post-mortem examination in a house not far from the Hospital. "The man who kept the shop had only one leg, and made use of a crutch. His wife had died after an obscure illness, so that the medical man in attendance asked for a post-mortem, and we accompanied him. Hilton found an abscess of the liver, and immediately exclaimed, "This did not come of itself; she has had a blow with that crutch," which he had observed on entering; he then found a broken rib, and it turned out that in all probability Hilton's surmise was correct."[1] It was perhaps owing to Hilton's sharing the work in the post-mortem room that, as the account in our Reports for 1841 runs, "the examination of the body of Sir Astley Cooper in the seventy-third year of his age was made on February 13th, 1841, at nine o'clock in the evening, thirty-two hours after death, by Mr. John Hilton, in the presence of Dr.

[1] 'Biographical History of Guy's Hospital,' Wilks and Bettany, p. 348.

Chambers, Dr. Bright, Mr. C. A. Key, and Mr. Edward Cock." It will be remembered that "shortly before his death Sir Astley Cooper expressed a wish that the appearances which should be presented on the inspection of his body might be recorded in the 'Guy's Hospital Reports.' He had particularly alluded to four points, the investigation of which he thought desirable,—a cured oblique inguinal hernia; a cured umbilical hernia; some suspected indications of phthisis in his youth, and an inability to sleep while lying on his left side."

In 1845 or 1846 Hilton was made Lecturer on Anatomy, an appointment which he held till 1853. Anatomy lectures at this time were given six days a week, and at two in the afternoon. Anatomy had not long been separated from Physiology, Sir William Gull being, we believe, the first lecturer appointed separately for Physiology at Guy's. Sir B. Brodie had written largely on subjects which would now be called physiological. Sir J. Paget was lecturing on physiology. It was probably this fact, that the two subjects had not been long divided and placed separately in the hand of physicians and surgeons, that led on Hilton more and more to take the line of Teleology in his lectures on Anatomy, and many other points combined to lead him in the same direction. Teleology at that time, and for long after, strengthened by the publication of the Bridgewater Treatises, reigned supreme in the London hospital schools. At Edinburgh, Knox was in the habit of delivering, as part of his course, lectures on comparative anatomy and ethnology, and had been eloquent, though superficially so, on transcendental or philosophical anatomy; but this most famous teacher, owing to his having received bodies from the murderers Burke and Hare, within a few hours of the murders had been exposed to the risk of Jedart justice, and though exonerated by the confession of Burke and the investigations of a committee, had, after a series of brilliant successes, like his idol, met with his Moscow and Waterloo, and his unique position in the extra-mural School at Edinburgh had been taken by Professor Goodsir as University Professor.

On the subject of Hilton as a teacher of Teleology in Anatomy we may quote from an article unsigned, but cer-

tainly by Dr. Moxon, in the 'Guy's Hospital Gazette' for December, 1878: "With Mr. Hilton, the things he saw became matters of living knowledge, for he was always upon the question, 'Why is this thing so? What is the end or purpose which it serves?' His was not the modern question, 'How has it arisen? by what correlation of conserved purposes in vast lapse of time did it evolve itself out of nothing particular?'—a question which rots young surgeons. Hence his power as a surgical teacher: hence he was not drawn aside into vapid 'biology.' He never showed the least taint of troglodyte philosophy, the amateur professorship of which seems now to be epizootic amongst the very highest classes. To him anatomy revealed designs and objects which he could further in his surgical efforts, not casual adaptations of a blind and brainless universe, begetting correlated brainlessness in the curiosity-mongers who collect a row of other specimens to link with themselves into a chain of being downwards, and to resolve into primeval mud the mystery of human existence. Mr. Hilton never lent his powers to this noble achievement."

Hilton, as a lecturer, had the art of saying things which had never been said before, the great secret of interesting students, putting the trite and oft-told facts of Anatomy in a totally new light. Words from his lips, "You perceive, gentlemen," or "You observe," as he pointed to some detail in the relations of the sphenoid, some point in the distribution of the branches of the subclavian, or in the origin or ending of the vagus, was sure to be emphasised by some original remark, not couched in any brilliantly flashing words, but in sober, blunt English, and based on his own observation and his own experience.

To those who knew not Mr. Hilton, to whom his name is but a name, the following passages will serve to prove how habitually he made dull and matter-of-fact details interesting, and how fascinating his Anatomical Lectures were. They will, further, give proof of his precision, his thoughtfulness, and how, as day by day he stood in the Anatomical Theatre, he threw floods of light upon his subject, and how, in his hands, facts which with other

teachers would have worn a monotonous Dryasdust aspect became welcome and specially instructive lessons.

It is part of his course on the arteries, and he is alluding to the great precision which marks the supply of arterial blood to some parts of the body. He asks, "Why should not the whole of the rectum be supplied with blood from the inferior mesenteric artery? At first thought, there appears to be no reason why that artery should not carry the blood downwards to all the lower part of the rectum, as well as to the upper; yet it is not so. The lowest part receives its arterial supply almost exclusively from the pudic artery. A few branches may be traced from the inferior mesenteric and middle sacral to join the pudic, but the chief supply is derived from the pudic arteries, which thus bring the neck of the bladder, the perinæum, and the urethra into *intimate structural association* with the rectum. As I think this kind of definite distribution of arteries is a point not sufficiently dwelt upon, allow me to remind you of a few illustrative instances observed in the human body. Take, for instance, the cœliac artery, which is distributed to the organs of digestion, and nothing else. It supplies the liver, stomach, pancreas, spleen, and part of the duodenum; that is all that it does. As these are all organs connected with the process of digestion, the cœliac artery might well be called the "digestive artery." Take another illustration. The os hyoides, placed between the larynx and the pharynx, is, functionally and structurally, associated with both. Does not anatomy tell us that the os hyoides receives a branch from the superior thyroid artery, and a branch from the lingual artery on each side? Is not the os hyoides connected with the functional and structural integrity of both the larynx and the tongue, intimating the reason of the double source of arterial blood for its growth and nutrition? There is a disposition on the part of anatomists to think and to teach that nerves are distributed with designed accuracy, but that there is very little design in the distribution of the arteries. I opened up this subject many years ago, when endeavouring to explain to the students of Guy's Hospital the purpose of the division of the subclavian artery into its numerous

branches. I pointed out that the branches distributed from the subclavian trunk, apart from its continuation to the upper extremity, are distributed with one simple purpose, viz. to supply all the parts concerned directly and indirectly in the process of respiration—that is the simple object of the distribution of the subclavian artery. Thus the vertebral, besides supplying other parts, is distributed to that portion of the spinal marrow from which the phrenic, spinal accessory, and posterior thoracic—all important nerves of respiration—take origin.

The internal mammary supplies the sternum, cartilages of the ribs, origin of the pectoralis major, phrenic nerve, diaphragm, and the upper half of the respiratory portion of the abdominal muscles.

The superior intercostal artery goes to the first and second ribs. Now the first is the most important of all the ribs in the respiratory function, as it forms the fixed point for the action of the intercostal muscles in elevating the chest.

Of the branches of the thyroid axis, the inferior thyroid builds up the trachea, a tube essentially connected with respiration, and sends a branch upwards (ascending cervical) which supplies and nourishes the phrenic nerve, and constitutes, in fact, an ascending comes nervi phrenici. The transverse cervical supplies the trapezius and the posterior border of the scapula, with the muscles attached to it, all of which may be considered accessory to respiration. The supra-scapular supplies the clavicle and scapula, both of which are rendered respiratory by the attachment they give to accessory respiratory muscles."

Even a better instance of Mr. Hilton's ingenious way of reasoning from anatomical facts, and finding a purpose in every point of detail, even a clearer example of Hilton's way of looking upon Anatomy not as a heap of facts more or less isolated and detached, but as teaching, each and every one, most important object-lessons will be found in the way in which he lectured on the internal maxillary, or, as he called it, the true " masticatory artery." He is speaking of its branches, and first of the middle meningeal and its course to the interior of the skull. " If it were only for the sake of a supply of a certain quantity of blood to the dura mater, the

special intra-cranial arteries, viz. the two internal carotids and the two vertebrals, would be amply sufficient for this purpose, and there would be no necessity for the entrance of those streams that are derived from vessels supplying parts on the exterior of the skull. There must, therefore, be some design in such an arrangement. The essential purpose of the internal maxillary artery is to build up or nourish those parts directly necessary or accessory to the process of mastication, so that it may be fairly called the "masticatory artery." From this trunk is derived the middle meningeal artery, which, whilst ramifying on the dura mater, gives branches outwards, that supply the squamous portions of the temporal, the greater wing of the sphenoid, the frontal and the parietal bones—those bones, in fact, which contribute to form the temporal fossa, the chief origin of the temporal muscle, one of the most important agents in mastication. The middle meningeal artery, therefore, may be said to be sent by the internal maxillary into the interior of the cranium to build up, and afterwards to nourish that portion of its extent which, in giving attachment to masticatory muscles, is rendered subservient to the masticatory function. So fully, indeed, is this intention carried out, that a small artery given off from the middle meningeal, or the trunk of the internal maxillary itself, enters the cranium by the foramen ovale apparently for the express purpose of furnishing nutritive material to the third division of the fifth—the nerve that supplies the muscles of mastication. Now although the intra-cranial portion of the third section of the fifth is situated in such close proximity to the entrance of the internal carotid into the skull, yet it is not supplied by this vessel, but, as we observe, receives a distinct offset from the masticatory trunk, so that its nutrition may be maintained in the closest possible relation with that of the other structures administering to the same function."

His Lectures on the Cranium, some notes on which were reported by Dr. Pavy, and thus fortunately preserved, show Hilton's power of making "dry bones live." It is much to be regretted that other parts of his course have not been thus handed down to us by equally careful listeners.

Hilton's description of the ridges and elevations of the

inner aspect of the cranium—their thickness, to enable them better to conduct vibrations—their course, curved so as to weaken those vibrations, and definitely directed to certain points in the base, such as the clinoid processes, whence they are transmitted to the adjacent cerebro-spinal fluid, or lost in the membranous texture of the dura mater, where they terminate without injury to the easily lacerable cerebral tissue, these passages are well known. We will quote one other, which proves how, even in ossification, that *bête noir* of a student, in Hilton's hands, and in his reflective, sagacious mind, complexities that seem merely vexatious, details that appear meaningless, are all shown to have a meaning, and to influence, most advantageously, the organs with which they are connected.

He takes as his text the development of the sphenoid, and the numerous and intricate changes in the direction and arrangement of the whole cranial and facial bones which its growth and final completion brings about.

"The sphenoid bone forms the centre, around which all the other bones, both of the cranium and the face, are developed. It is truly and literally, indeed, a wedge, as its name implies; and thus impacted or wedged in amongst all the other cranial and facial bones, its progressive development spreading its different processes out in all directions, plays a most important part, not only in determining the adult configuration of the skull, but in adapting the final conformation of the organs of the face to the increasing perfection of their associated functions. The mouth, nose, orbit, and pharynx are all more or less directly influenced, and contemporaneously rendered more perfect in form, by the complete development of this bone. All those harmonious changes, also, which accommodate the framework of the masticating apparatus to the increasing number and size of the teeth,—all these, I say, are brought about in a manner I shall presently point out by the progressive development of this bone. By the student the sphenoid bone is regarded as the most complex bone of the body; and precisely in proportion to the anatomical complexity it presents are the variety and importance of its functional or physiological connections.

"The primary intention of the development of the sphenoid seems to be chiefly with reference to the masticatory function, but in the changes that it produces in the direction of the cranial and facial bones it may not unaptly be compared to the scaphoid bones of the carpus and tarsus, for in its growth and final development it effects for the cranium and face precisely the same object that these bones effect for the hand and foot.

"The scaphoid is the last of the tarsal bones to become ossified, and in its growth it not only widens or spreads out the foot, but lengthens the ankle, and increasing its elasticity, thus completes the efficiency of the whole as an organ of support and locomotion. So, again, the scaphoid of the hand, with the trapezium and trapezoid, forms the latest of the carpal bones to be ossified and completed, with the exception of the pisiform bone, that seems to act rather as a mechanical defence to the ulnar nerve and artery than to be developed like the other carpal bones in relation to the movements of the hand. During early life, the thumb is as nearly as possible parallel to or on a level with the fingers; but, as age advances, and the development of these carpal bones proceeds on towards completion, the thumb is pushed forwards and outwards so as to be placed more opposite the fingers, thus materially increasing the efficiency of the hand as a prehensile organ, and adapting it for those delicate and varied operations for which it is destined in the adult human subject.

"The striking alteration in the direction of the petrous bones, which is in wise harmony with changes taking place in other parts, is effected by the full expansion or complete development of the spinous processes of the sphenoid. As the petrous bones enlarge, each spreading out in all directions from its own centre, the two would necessarily have a tendency to approach each in the median line, and thus to crush the intervening body of the sphenoid if they retained their original direction. But at the same time that the petrous bones are elongating the spinous processes of the sphenoid are growing and proceeding in a backward direction, and, being inserted into the receding angle between the squamous and petrous bones, they push the whole temporals

backwards and alter the direction of their petrous portions, which, now running more obliquely forwards, can find space for elongating without encroaching upon the central parts of the base.

"Besides altering the direction of the petrous bones the development of the spinous process of the sphenoid, fitting in between the squamous and the petrous, or the masticating and auditory portions of the temporal, acts on both alike, and pushes the whole bone backwards and outwards, thus giving much increased space for the full expansion of the frame-work administering to the masticatory function. It is to the temporal bones that the glenoid fossæ belong, and these articulate with the condyles of the lower jaw. The temporal bones, therefore, in being impelled backwards and outwards, carry with them the glenoid cavities, and in this way not only give a greater area laterally to the pharynx, but also increase the capacity of the mouth both laterally and in an antero-posterior direction. The object of such additional capacity is sufficiently clear, viz. to supply the necessary space required for the increasing size and number of the teeth. We thus see how the changes effected by the evolution of the spinous processes of the sphenoid forms one of the illustrations of the relation that the development of this bone bears to the perfection or completion of the masticatory apparatus.

"The temporal bone, on being thrust backwards, carries with it the parietal which overlaps the portion of the brain associated with the mental faculties, which, like the sphenoid (the bone that effects this change), is late in arriving at a complete state of evolution or development. The union between the temporal and parietal is exceedingly secure; the one bone being, as it were, locked into the other by an alternate overlapping or bevelling of their opposed surfaces of contact. Were it not for such a provision of security, the temporal bone, on being thrust backwards, through the agency of the spinous process of the sphenoid, would glide underneath or be displaced from the parietal, instead of carrying this bone backwards with it."

It is difficult to make selections where all is so excellent. One more extract must suffice to show Hilton's characteristic

way of dealing with a bone, taking it point by point, finding in each detail an opportunity for throwing one more ray of light, and finally, when the description was completed and perfected, leaving his hearers with the feeling that Anatomy had never been made so interesting as it was at Guy's at that day.

"The vomer is a flat plate of bone which occupies a median vertical position in the nasal cavity, and which, through the influence of the sphenoid, is mainly conducive in determining the final configuration of the nose and mouth. By its inferior border it articulates with the upper surface of the hard palate, while its superior border is adapted for the reception of the rostrum and the azygos process projecting from the under surface of the body of the sphenoid, which, thus fitting into the vomer, forms an exceedingly secure and efficient articulation between these bones. From this anatomical relation we can perceive how the development of the body of the sphenoid, expanding into the sphenoidal cells and spreading out in all directions, is capable of exerting an influence through the medium of the rostrum which ploughs the vomer onwards in a downward and forward direction, and so alters the relative position of the neighbouring parts, in anticipation of those higher functional attainments acquired with adult age. The inferior border of the vomer being firmly affixed to the hard palate, its advance necessarily has a tendency to push the superior maxillary bones in a direction downwards and forwards, so as to increase the capacity of the mouth from before to behind, thereby giving room for the development of the molar teeth, and at the same time to deepen the cavities of the nasal fossæ, in this way affording space not only for the complete development of the turbinated bones and the extension of the olfactory surface, but also for the more ready ingress and egress of air through the nasal organ. By its connection, anteriorly, with the central lamella of the ethmoid, its advance also pushes forward this plate of bone, which in its turn elevates the nasal bones, and likewise leads to the formation of the frontal sinuses"—*i. e.* by the advance of the anterior table of the frontal bone, with which the nasal bones are connected.

But it was probably in Hilton's Anatomical Lectures on the nervous system that his naturally great, though somewhat rough acumen, his shrewdness as a thinker, his great good sense, and his habit of submitting what he taught to the test of experience were, one and all, most brilliantly conspicuous.

In his descriptions of the brain and spinal cord he was a head and shoulders in advance of other writers. Far from being content with giving his hearers mere topographical details, he strove to fit every point by some practical application, or some original, thoughtful, and suggestive view of his own; and if his theories were sometimes fanciful and speculative, they were always ingenious and stamped with originality. Vast as have been the advances of late years in this field, Hilton's truly scientific spirit led up to the point on which we stand at the present day.

The cerebro-spinal fluid is an excellent instance of Hilton's method of throwing new light upon everything he touched on. Instead of dismissing it with a few words as to its chemical and physical character, he devoted a whole lecture to it. While lecturing on Anatomy in 1844, he had had his attention drawn to hydrocephalus, and had found in nearly every case that the cerebro-spinal aperture between the fourth ventricle and the spinal subarachnoid space was completely occluded by dense membrane. The following is a good example of Hilton's keenness to recognise the importance of anatomical and pathological facts, his ingenious way of piecing them together and reasoning from them, and his application of these facts to daily practice:

"I said previously that I should be prepared to detail another case of occlusion of the cerebro-spinal aperture of considerable interest. The gentleman who was the subject of this disease died at the age of thirty-four. On the day of his death he had visited the Crystal Palace. He had been seized with vomiting in the morning, and again about two hours before he reached home. He walked from the Crystal Palace (a mile or so), and when he entered the house he staggered, and said he felt giddy and oppressed. He was placed on a bed, and cold was applied to his forehead; but he died in a very short time, with stertorous breathing. I examined the body, assisted by Dr. Wilks,

and this is the report of the post-mortem examination :—
'Cadaveric decomposition was advanced, except within the brain, which presented in this respect a remarkable contrast to the other parts of the body. The convolutions of the cerebrum were large, and their surfaces flattened by pressure from within.'

"These convolutions were few, suggesting, perhaps, that the condition of the brain was a persistent state of a very early development of the convolutions; for although the person was full grown, and thirty-four years of age, the convolutions were large and few.

"The brain structure was healthy, neither it nor the membranes containing any tubercular deposit. On carefully opening the lateral ventricles, they were found to contain at least four ounces of clear cerebro-spinal fluid.

"Regarding the character of this fluid there can be no doubt. It was carefully examined. The ventricles were greatly enlarged, and all the receding and projecting angles of the boundaries and cornua were lost or subdued. The foramina of Monro were rounded, and capable of admitting a large quill, not quite a third of an inch in diameter. The fourth ventricle was greatly dilated in all its directions, showing well its lateral cornua, which support the pneumogastric, auditory, and other accompanying nerves. The cerebro-spinal opening between the under surface of the cerebellum and the upper surface of the medulla oblongata was completely closed by a tolerably dense membranous structure, which formed a kind of pouch, projecting downwards, and showed the direction of the fluid tension upon it to have been from above to below. This was clearly seen when looked at either from the ventricular aspect, or below, from the spinal aspect.

"The details of the history during life present an apparently perfect fitting in of the symptoms to the pathological anatomy. But I wish it to be understood that this account has not been 'cooked,' as it is termed. It was not made up for the purpose of agreeing with the facts previously stated, but was drawn up by the brother of the gentleman himself, who is not a professional man. 'This gentleman always had delicate health. The nails of his fingers were peculiar in

form, the last phalanges being very short, and the nails inserted like two seeds into the cuticle.'

"I stop at this point to remark that here apparently we have evidence of a congenital error in the development of the fingers, and this inclines me to the conclusion that probably the occlusion of the cerebro-spinal aperture was also congenital.

"As a child he was active and wiry, but very irascible in temper. He had a fondness for intellectual pursuits; also for turning light fancy work, and for gardening; and a great dislike to the excitement, and especially the noise of London. Indeed, at the age of sixteen he had a severe nervous illness with great depression, brought on by application to business in the City; it ought, however, to be mentioned that his occupation was not at all intense, and nothing would have been thought of it by ordinary men.

"Now this is worthy of note, because in this instance there was no possibility of the cerebro-spinal fluid escaping from the interior of the brain. Whatever pressure might be made by mental occupation or physical exertion upon the internal parts of the brain in the medulla would be morbidly manifested, in consequence of there being no possibility of escape of cerebro-spinal fluid, and because there was no aperture to allow of it.

"His food was always of the simplest kind. Even tea, coffee, and cocoa seemed to affect his head and derange his stomach; and he had an instinctive dislike to alcoholic stimulants of every kind. Though subject to coldness of the extremities, he could not bear a warm room, because it made him feel faint. Both winter and cold always affected him injuriously. He said himself he only half lived in the winter; he seemed torpid, and would drop into a deep sleep, from which it was often difficult to rouse him at bedtime, when he seemed scarcely to know where he was. Excitement frequently brought on an impediment in his speech. For many years he was subject to headache, derangement of stomach, and occasional deafness. His pulse was extremely weak, irregular, and slow, from fifty to sixty beats per minute. He was thought to be suffering from heart disease. We have here, if we may so term it, a most important and

valuable living experiment. Here is a man who cannot bear any accumulation of blood in his brain, because there is no possibility for the compensatory fluid to escape from the pressure or state of congestion of the organs in the interior of the skull. I conclude that we find the cause of the stomach affection and the deafness in the great distension of the fourth ventricle, and the consequent pressure upon the pneumogastric and auditory nerves on its floor.

"If I were to select this opportunity to enlarge upon the apposite circumstances of this case, perhaps I might do so at too great length. I will merely observe that I think it an important and interesting experiment in reference to the circulation through the brain, the administration of food and stimulants, and various other matters. His brain could bear no fulness of blood, no increase of size, because the cerebro-spinal fluid could not escape from the interior of the brain. He could not take wine, beer, or spirits, and could bear no muscular exertion. The congestion of the brain produced vomiting by pressure upon the medulla oblongata, exerting its influence upon the pneumogastric nerve. He died from pressure upon the medulla oblongata—that pressure which could not be obviated."

On account of our space we must allude more briefly to some of the other more striking points in Hilton's anatomical teaching. It is easy for generations who have arisen since to forget who first it was who, by a wealth of anatomical knowledge garnered by the labours of his earlier years, first placed on a firm foundation facts which seem trite and familiar now.

To take the subject of "The Precision of the Nervous Supply to Muscles," other anatomists, *e. g.* Swan, had published valuable books on the distribution of nerves; it was Hilton who first made Anatomy Lectures interesting as well as valuable by the accuracy with which he traced each nerve, and by the wealth of illustration with which he clenched and drove home every point in their distribution in the memories of those who listened to him.

To use favourite words of Hilton, he used to "put the subject in the plainest practical way" when lecturing on the Nerves of the Scalp. He first drew attention to the

importance of distinguishing the different areas according to their nerve-supply, using diagrams far from ornate, brilliantly dauby in colour, but sufficing to him because they illustrated the points on which he was going to dwell. Thus pain at the back of the head and behind the ear might be due to disease of the upper cervical vertebræ affecting the great and small occipital nerves, pain in the lower part of the ear to a suppurating cervical gland lying close to the great auricular; pain in the auditory canal itself to something which concerned the auriculo-temporal nerve.

At another time, in the lectures on the fifth nerve, this distribution of the auriculo-temporal to the meatus of the ear used to lead to the association of earache and toothache, and thus a little later to unilateral furring of the tongue. This was one of Hilton's pet subjects, and one which he claimed as peculiarly his own. In the somewhat sententious *sesquipedalia verba*[1] in which he rather delighted, it was a "functional and unnatural deterioration depending upon nerve influence." In other words, this condition of the fur is one of the effects which disturbance of the nerve-force may produce on the nutrition of a part. Fifty years have gone by since Hilton first had an opportunity of clearing up a case of this kind. It is interesting on another account, as showing the zeal with which he followed up his cases, seeking the information which is given alone in the silence of death. "On December 17th, 1843, I examined with Mr. Blenkarne, a surgeon in the City, the body of one of his patients who had died with disease of the brain and spine. She had suffered from intense pain on the left side of the head. She had also, during her life, a tongue furred on the left and scarcely at all on the right side. It became, therefore, an important point to clear up, what was the probable cause of that condition of the tongue. Upon a very careful examination of the head, on turning up the dura mater from the anterior part of the petrous portion of the left temporal bone, for the especial purpose, we exposed the Gasserian ganglion, or the ganglion of the fifth nerve. We there

[1] The "seven-leagued words" of Dr. Rochecliffe in Sir W. Scott's 'Woodstock.'

found what might be called a scrofulous deposit upon the convex edge of this ganglion, involving the second division of the fifth nerve more than the third, but still involving all more or less. Here, then, appeared to us to be the probable explanation of the tongue furred on its left side. In 1844, when delivering some lectures upon the nerves at Guy's Hospital, I mentioned the case, and one other that I had seen before, the cause of which I had not distinctly proved. After the lecture one of the students said, 'Look at my tongue; it is furred on one side, and I cannot get rid of it.' He had a decayed and painful tooth—the second molar in the upper jaw. That tooth was, by my advice, removed, and after a short time the fur on the tongue entirely ceased. Some time afterwards a case of fracture of the skull was brought into Guy's Hospital, where the fracture ran across the foramen rotundum, containing the second division of the fifth nerve. In that case the man had a furred tongue on the same side, indicating a probable relation of cause and effect."

One of the most observant of living surgeons, and one who, perhaps, more than any other has added to the knowledge of his own and future generations—Mr. Hutchinson—has put forward ('Lond. Hosp. Reports,' vol. iii, p. 392) quite a different explanation of these unilateral furrings of the tongue, viz. that the side of the face which corresponds to the furred half of the tongue is but little used in mastication, and hence no renewal of the epithelium of the tongue takes place by friction of the food on that side. In Mr. Hutchinson's words, "One-sided fur of the tongue is, I believe, caused simply by the circumstance that its subject is accustomed to eat on only one side of his mouth. . . . It is often present where there is mere loss of teeth, no toothache or source of irritation. I therefore cannot agree with the hypothesis which supposes it to depend upon reflex disturbance of nutrition through the lower branch of the fifth. It is quite true that it occurs in its most exaggerated form where there is toothache, but this is precisely the condition in which mastication on the affected side would be most sedulously avoided." In answer to this, while we may point out that there are cases to which, owing to the absence

of toothache or any irritation, Mr. Hilton's explanation will not apply, it is quite clear that the one which Mr. Hutchinson would substitute will not account for those cases where, quite apart from toothache, there is a distinct source of irritation, deeply situated within the cranium, as in two of the cases instanced above. Nor will it meet those cases in which the furring of the tongue is not unilateral over the distribution of the lingual gustatory, but limited to the posterior part of the tongue. For Hilton also showed that in certain cases of disease of the tonsil, *e.g.* inflammation, syphilitic ulcer, &c., a region which is supplied by the glossopharyngeal nerve, the posterior part only of the tongue (which is supplied by the same nerve) is furred also.

Before we leave Hilton, and the way in which he made the cranial nerves interesting, we may refer to one more instance, not only because it is a fresh example of his power of tracing cause and effect, and throwing light on obscure places, but because with it is linked another name—the name of one greater than Hilton, and one who, in spite of occasional differences, was one of Hilton's most staunch admirers.

"In a preceding lecture I mentioned that a professional friend of mine had suffered from a condition which bears on the subject of the influence of nerves on the structures supplied by them. That friend, unfortunately, is no more. It was Dr. Addison to whom this happened. Some years ago Dr. Addison had a very offensive discharge from one external auditory meatus, which annoyed him very much, and below the ear was a small gland, enlarged. He had tried various remedies, and had gone, I believe, to some surgeon who attends specially to the ear; but, as far as I could learn, no good resulted from any of their applications. Upon examining the ear from which the offensive discharge proceeded, I found a slight ulceration upon the floor of the auditory canal. On discussing the question between us, we came to the conclusion that the ulceration probably depended upon a diseased molar tooth in the lower jaw on the same side. We had that tooth extracted, and in a very short time the ulcer healed, the discharge disappeared from the auditory canal, and as soon as that ulceration was cured the

enlarged gland subsided. Here the true cause of error was remotely situated from one of the most prominent symptoms, namely, the enlarged gland in the upper part of the neck. In this respect the case is of great interest."

It was just the same with the nerves in other parts of the body ; for every one Hilton had some ingenious suggestion to make, which illustrated from his favourite Teleological point of view some detail in the course or distribution of the nerve, or some case to quote in proof of the importance of a precise and accurate knowledge of Anatomy. To give a few instances :—" As a rule, nerves enter the muscles where they would be most secure from pressure ; and it is curious to observe how careful nature has been in this respect to guard one of the most important nerves of the body. The phrenic nerves (our lives hang upon these threads), after passing through the chest, traverse the diaphragm, and distribute their branches upon the under surface of the muscle, and are so situated that they cannot be compressed during respiration. If they were distributed upon the upper surface of the diaphragm, where there is a constant and forced contact between the base of the lung and the superior aspect of the diaphragm, and especially so during a retained inspiration, it is obvious that these filaments of the phrenic nerve would under these circumstances be exposed or subjected to compression, and the action of the diaphragm would be dangerously interfered with. The nerves, however, are distributed upon the under or concave surface of the diaphragm, the whole tendency of gravitation being to remove the liver, the stomach, and the spleen away from them, so as to enable the nerves to carry their influence to the diaphragm unmolested."

The two following cases are classical, though at the present day their explanation seems obvious enough, and with the advances of modern Surgery the cause could in each case have been dealt with. " Elizabeth B—, aged 47, came under my care at Guy's Hospital in February, 1853. She was of healthy aspect and regular habits. About a year before, she felt numbness in the extremities of the third and fourth fingers of the left hand, with pain in the same parts, especially increased during the night. The fingers gradually

became enfeebled and lessened in size. In a short time the ends of the above-named fingers began to appear discoloured, while the numbness and pain increased. She had no particular treatment for this, and, as mortification was impending, the surgeon advised her to come to the hospital. On admission the third and fourth fingers were found to be gangrenous. No pulse was perceptible in the radial, and only a very slight one in the ulnar artery. On examining the case to ascertain the cause of all this, I found upon the first rib an exostosis pressing upon the subclavian artery so as to prevent any blood from passing through it. The exostosis was pressing upon a nerve also, which turned out to be the ulnar nerve. I pressed my finger upon the nerve overlying the exostosis, and immediately the patient experienced additional pain in the little and adjoining fingers."

"Some years ago a sailor came to Guy's Hospital under the care of the late Mr. Key, having had a bad fracture of the lower part of the radius. There was no surgeon on board the ship; the fracture was not properly set, and in consequence the callus pressed severely upon the median nerve above the wrist. The man had ulceration of the skin upon the thumb, the forefinger, and the middle finger. The hand was purposely flexed, and all the tension was taken off the median nerve, and the ulceration got perfectly well; but as soon as his hand was allowed to be freely used and extended again, and the pressure of the callus permitted to exert its influence upon the median nerve, the ulceration reappeared. He could not pursue his occupation as a sailor, but it was not thought right to submit him to any remedial operation, which might place his hand in danger; so he preferred remaining as he was, subject no doubt to a recurrence of the ulceration to which I have alluded."

We have mentioned Dr. Addison's name: before leaving Hilton as a Lecturer on Anatomy it may be mentioned that that great physician did his surgical colleague honour in a way which is probably quite unique. For many years, when Hilton reached that part of his course relating to the brain, Addison used to attend,[1] a chair being placed for him just within the door by which the lecturer enters from the

[1] Dr. Constantine Holman.

Museum. Some have thought that this was partly due to Addison's knowledge of Anatomy being deficient. This is possible. Addison took his degree at Edinburgh in 1815: at this time the Chair of Anatomy was still in the hands of the Monros. Monro (Primus) and his son (Secundus) had passed away after laying firm foundations on which the Anatomical School of Edinburgh was to flourish later; in 1808 the third Monro was beginning his long and somewhat tedious reign, and Barclay had not long begun to show that zeal which was to make him famous as an extra-mural teacher. But the chief excuse, if one were needed, for the most eminent physician of his day wishing to refresh his knowledge of Anatomy lies in the fact that at the time Addison was at Edinburgh the supply of subjects was notoriously unequal to the demand. Owing to the population of the capital being comparatively scanty, and the fame of the Edinburgh school, the demand was so much greater than the supply that Dr. Barclay, in 1806 and later, was sending to London for subjects—a precarious and necessarily costly speculation. Dr. Lonsdale tells us that "the trading smacks plying between London and Leith were often detained by adverse winds, and the boxes of mortalities marked 'perishable goods' seldom reached their destination in a satisfactory condition." So, too, with regard to the other source of transit, that by coach. The art of packing and the use of anything approaching to antiseptics seem to have been utterly wanting. The same authority writes, "The danger of discovery was greater in the towns where the coach stayed for the night. Several inquests were held at Carlisle on bodies thus detected, and meant for the Edinburgh school. The decision of the juries on these occasions—'found dead in a box'—used to amuse the Scottish jurists, and naturally threw discredit on the proceedings of all 'crowners' 'quests.'" If Addison's knowledge of Anatomy was deficient there was abundant excuse, but we suspect that there was another reason. Addison, as we have seen, had been a patient of Hilton's: there was much in this leader of men to make him respect the sagacity, the vigour, and the other sterling qualities of his younger and surgical colleague. If Addison, with his immense reputation, and his position, in Dr. Wilks' words, "as the leading light

at Guy's, much superior to what Bright could ever claim, and equal, if not greater than that of Sir Astley Cooper," could thus find it worth his while to attend Hilton's lectures on Anatomy, we can understand how it was that when 2 p.m. came, students of all years were seen flocking to the Anatomical Theatre. Dr. Pavy recalls how on Tuesday and Friday at this hour, when Hilton left the operating Theatre to lecture, the Theatre emptied to follow him.[1]

We turn now from Hilton as an Anatomist and Teacher of Anatomy to his work as Surgeon and Teacher of Surgery. In 1845 he was elected Assistant Surgeon at Guy's. The surgical staff at this time included Morgan, Key, and Bransby Cooper as surgeons, and Callaway, Cook, and Hilton as assistant surgeons. By this time Hilton had had eighteen years' work in the Medical School. There is an impression in the minds of some that Key disliked Hilton, and kept him back. There is probably some truth in this. From 1830 and onwards Key was the first surgeon of the day; and this, together with the fact that in 1824, only three years after his qualifying, and when only thirty years old, Key had been appointed full Surgeon at Guy's, will account for much of his haughty, imperious way, and his habitual self-absorption in the success of his own paths in life. However this may have been, we shall see that Hilton, a little later, could speak very generously of his somewhat arrogant senior. Hilton at once devoted himself to the out-patients. He was well aware that he was behindhand in purely professional knowledge, and this very largely through no fault of his own. During his eighteen years' work at Anatomy and Pathology he had had no care of cases and no opportunity of practical work. At this time, be it remembered, the increasing population of London had not brought about the formation of those smaller hospitals, of which so many exist now, and at which it is possible for juniors to gain valuable experience, while waiting. Hilton's previous work had been almost entirely on purely scientific subjects. And perhaps one other fact should be mentioned if this memoir is to have

[1] It must be remembered that at this time students in their first year, owing to the shortness of the "curriculum," and the much smaller number of lectures, used to attend on the surgical practice of that day.

any of the honesty and straightforwardness which were largely characteristic of its subject. Hilton was not made Assistant Surgeon till 1845. We have seen that the models which we owe to him and to Mr. Towne were finished by 1838. In the years which intervened between the above dates Hilton was, perhaps, a little slack. Some of his early freshness and zeal had passed away with the fruits it bore, for in 1839 he had attained to the high honour of F.R.S. The truth is that certain changes which have taken place in the suburbs will account, in a measure, for any "neglect" of "purely professional work" (Dr. Wilks) for which Hilton himself may have been responsible at that time. He had one or two friends amongst the resident Fellows of that day in Dulwich College. This foundation then consisted of the old three-sided quadrangle, the picture gallery (originally collected for Stanislaus, king of Poland), four resident Fellows, a warden and chaplain; the present Dulwich College School, which did not start in its new buildings until 1870, being represented by Alleyne's original free school. The rising grounds of the surrounding manor, with which every one is familiar, were at that time richly wooded, instead of being thickly studded with villas. Dulwich was an isolated Surrey village, and there is no doubt that Hilton availed himself a good deal of the privileges which his friends at the College would give him of good dinners and rabbit shooting in the woods around.

However this may be, when appointed Assistant Surgeon Hilton must have made up for any lost time. Here, too, the scene as well as the actors have been greatly changed. The New Buildings, or Hunt's House, with its north and south wings, did not exist. Out-patients when Hilton was appointed were seen in a wretched room below ground situated on the east side of the two quadrangles through which the colonnade runs, and, being under ground, reached by a staircase. The part of the old building now occupied by the surgery and dresser's rooms was then the taking-in room, casualty room, and steward's office.

Hilton had not long to wait as Assistant Surgeon; in 1849 his promotion came to him in a somewhat tragic way. On August 22nd of this year Mr. Key was seized with symptoms

pointing to cholera while seeing patients at his house, and he died the next morning, after only twenty hours' illness. Hilton's promotion to the vacancy thus made is announced in the 'Lancet' of September 13th.

Two stories have come to us about this event, the one showing how unexpected was the vacancy, the other the outspoken honesty of Hilton's mind.

Dr. Constantine Holman relates that one day, a few weeks before Mr. Key's death, he, in passing along the colonnade, stopped to pay his respects to his old surgeon, and found him in a state of doubt as to whether he should obey a call to Frankfort to see one of the Rothschilds. The fee was 1000 guineas, but, as Key remarked, he never got up from his table in the morning without having taken ten or fifteen guineas, and these patients would not be missed only once, as many of them would come two or three times. "But what if some one else goes, sir?" "Ay, there's the rub." Key went. Hilton at the time was standing in the colonnade, a few paces off. Dr. Holman said in the course of the conversation, "Mr. Hilton has had a long time to wait for his beds." "Yes, and he will have to wait longer still," was the reply. A few weeks later Key was in his grave in St. Dionis' Churchyard, close to the back of his house in St. Helen's Place.

At the first school or staff meeting after the death of Mr. Key, Mr. Dobree, the treasurer (1848—1855), who, like many others, had quarrelled with that most eminent but somewhat dictatorial and arrogant surgeon, simply mentioned his death and his successor, and passed on to other matters. Hilton got up and spoke of the credit which Key had been to the Hospital and School, and then, warming *more suo* at what he considered an injustice, went on to say, "If no more notice than this is to be taken of Mr. Key, all I can say is that it's a d—d shame."[1]

[1] Dr. Pye-Smith. The ægis of my colleague's name will protect me from any accusation of habitual laxity of speech. Hilton never stopped to pick his words. And we must remember the habits of fifty years ago. Bob Acres had been long before his time—"The Rivals" was first acted in 1775—in his remark, "Damns have had their day." Hilton might have quoted great names as his excuse. Lord Melbourne, Prime Minister for the second time from 1835 to 1841, a gentleman in some of the best senses of the term, and a man whose name will always

Of Hilton as full Surgeon to Guy's we must try and give a sketch of the man first as a teacher in the wards and the theatre, and then as a lecturer on Clinical Surgery. To realise at all his power as a bed-side teacher, and his habit of getting information out of his cases, my junior readers must understand what a change Surgery has undergone since Hilton passed his twenty years as full Surgeon. Professor Lister had not introduced his great discovery, nor had Mr. Howse, as yet, gone to Edinburgh to return with those primitive and primeval implements of the Antiseptic Surgery of that day,—the putty, the irrigation tins, and the carbolic oil, which, introduced by him in 1870, not without much indifference and some ridicule, and constantly tended and fostered in his hands, have gradually developed into the Antiseptic Surgery as we know it at the present day at Guy's. Hilton's wards were Luke, Charity, and Cornelius—and the bedsteads are about all that have undergone no change. Primary union at that time was the exception, not the rule. Suppuration was not to be described (as by the modern ward clerk) "absent," or as "about a drachm in forty-eight hours," or as "a little sour;" but rather in the words of old Blucher when, after his fall in one of the final charges at Ligny, he had been brought round by copious potations of brandy (intended for use as a liniment rather than as a libation), and excused himself on embracing Sir H. Hardinge with the words, "Ich stinke etwas." The principles of drainage were then in their infancy, and but imperfectly carried out. Any ligature securing a vessel was left with its ends long. Dry lint and strapping were the first dress-

be held in honour for the pains which he took to initiate the Queen, when young and inexperienced in the years between her accession and her marriage, in the difficulties of constitutional government, was a notorious offender in this respect. The story runs that the Lord Ossington of that day called on Lord Melbourne with regard to some alterations he wanted made in the new Poor Law Bill. Lord Melbourne referred his visitor to his brother George, Under Secretary at the Home Office, and as much addicted to the use of profane expletives as himself. A few hours later, on their meeting again, the Premier asked Lord Ossington, "How did you get on with my brother?" "Very badly," was the reply. "He d—d me, and he d—d the Bill, and he d—d the paupers." "Well, d—n it," said Lord Melbourne, "what more could he do?" The witty sarcasm with which Sidney Smith rebuked this habit of the Premier is well known.

ings; later on, when suppuration set in, linseed poultices, lead and opium lotion, or a red lotion containing tartrate of steel. For fear of secondary hæmorrhage the flaps of a stump were not closed at once, but left to "glaze," and sutured later on in the wards.

The dressership to Hilton was considered as a Blue Riband.[1] In proof of this it is interesting to note that of our present staff the following were his dressers:—Dr. Pye-Smith, Dr. Taylor, Dr. Goodhart, Dr. Savage, Mr. Durham, Mr. Howse, and Mr. Davies-Colley. "Strong men were powerfully attracted by Hilton as a teacher."[2] Dr. A. Buck, of Ryde, writing of his time at Guy's (1863 to 1868) and his dressership to Hilton, says, "Much in surgery has changed, *toto cœlo*, since those days. I am not aware, however, of anything that Hilton taught me that has not been useful, or that has become obsolete."

This was due to the fact that Hilton taught on broad lines. He disliked names and conventional descriptions; the information he gave was all based on common sense and practical observation. His teaching of thirty years ago would have been equally valuable now-a-days. There was no conjuring of memories for particular theories; no discussion of useless details and possibilities, only to be forgotten at the next bed; and, above all, no fads.

Hilton relied upon his dressers not only for the treatment, but also for the reports of his cases. The ward clerks, not introduced until 1867, were a novelty that he ignored. The dresser was expected to have the report of each new case that fell to him ready by the time Hilton paid his next visit. If the report showed evidence of common sense and painstaking observation, words of satisfaction and occasional praise very likely followed. If the reverse, if there was any uncertainty in the dresser's mind as to what he might have observed, if proper names, obscure or theoretical terms were used, rebuke was certain, generally coupled with sarcasm, and, too often, with a sneer. Thus if, in Cornelius Ward, in answer to the question, "What have you got here, sir?" the dresser answered, "I think it's a Pott's fracture,

[1] Dr. Pye-Smith.
[2] Dr. Alpheus Buck.

Mr. Hilton," the reply was sure to provoke a storm. Hilton was no respecter of persons. As we have seen, his early education had been faulty, and he had—much too soon to be good for him—been obliged to trust to himself alone. "Vixere fortes ante Agamemnona" was scarcely enough an article of his faith, and the mention of the great St. Bartholomew's surgeon only led to an expression which relegated his name to undeserved obscurity as long as those dressers were in office, while the uncertain tone of the dresser's reply was likely to evoke "Think so! Do you think that you've hair on your head?" As was natural from the bent of his mind, Hilton was always wanting to know why this or that was done, in marked contra-distinction to carrying out any routine treatment. He was most minutely particular as to his cases being dressed properly—a point especially noticeable in his fractures. "Nothing approaching slovenliness escaped his eye, but provoked rebuke mingled with scorn, much dreaded by his dressers. Time after time," writes Dr. Buck, "he made me take down the same fracture, put up, as I thought, with the greatest nicety and care, because the patient complained of some pain, at the heel may be, or because Hilton could detect the line of fracture with his finger."

"That fracture is not in good position, sir; will you tell me why?" often fell from Hilton's lips, followed by questions on the origin and insertion of all the muscles concerned, their nerve-supply and grouping.[1]

It might truly be said of Hilton that he was *magnus in magnis*, and still more that he was *maximus in minimis*. However chronic and uninteresting, however trite and trifling seemed the case, he had the power of getting information out of it.[2] Thus it is told how one day, when he had finished one ward, and was waiting for something, he drew attention to a tedious case of necrosis, and pointed to the fact that the pus which was coming away pulsated.

[1] Mr. Couling.

[2] Dr. Wilks ('Biographical History of Guy's Hospital,' p. 348) writes, "Medical practitioners, meeting Hilton in consultation, were all agreed that, thoroughly as they thought they understood the case, this surgeon would come in and throw fresh light on it."

Turning to the students nearest to the case, he bade them notice how compression on the artery diminished the pulsation, while it increased when the vein was pressed on.[1]

Much has been said of Hilton's brusque way and rudeness to those with whom he worked—a matter to which we shall refer again.

The following shows that his sarcasm was not always of the cynical and harshly grating kind that it has been represented. We again gladly quote Dr. Buck: "On one occasion, when following him up the grand staircase, I was unconsciously whistling (being light-hearted in those days), when Hilton, suddenly stopping and parting the press of students following, beckoned me forward, saying at the same time, 'The band always goes in front, gentlemen.' On another occasion he noticed a student going round who was somewhat of a dandy, with an ornamental cane in his hand. Hilton, with much affected interest, requested the youth to let him look at the walking-stick. The owner seemed flattered by such notice, until, after looking it over with much minuteness and admiration, Hilton said, 'Pray, sir, what surgical use do you put this to?' At that time it was not unusual for some students to go round the wards in smart gloves. I remember, once, Hilton was examining a man *per rectum*—a case of fistula or malignant disease, may be—and, withdrawing his finger, he with much courtesy invited these elegantly gloved dandies to examine the case. As they, one by one, withdrew to the rear of the crowd, he said, 'Gentlemen, never miss any opportunity of examining the rectum you get; if you do, you will surely live to regret it.' These words impressed me greatly, and I always acted up to them. Some years after they stood me in good stead. I was newly qualified, and acting as *locum tenens* in private practice, when I was sent for to an old gentleman, very irascible and impatient, whose case I could not readily diagnose. He complained of constant tenesmus and pain in defæcation, which had come on suddenly and recently. Nothing was to be seen externally, such as piles, fissure, &c. Suddenly Hilton's words crossed my mind, 'Never miss an opportunity,' &c. I passed my finger, and found about two

[1] Dr. Pye-Smith.

inches up a large fish-bone jammed transversely across the gut, the removal of which cured the case."

It was to a similar kind of dandy, and perhaps about the same time, that a remark of Sir W. Gull referred. It was a Whit Sunday morning, bright with glorious sunshine streaming into the ward, and the physician was paying a Sunday morning visit to urgent cases. He came to a case of enterica, where the patient, in all the apathy of grave illness, lay flooded in a recently passed evacuation. An "exquisite," arrayed in all his Sunday best, drew back lest his outer man should suffer defilement. One of Sir W. Gull's sententious and somewhat mystic aphorisms promptly followed: "God's sunshine, gentlemen, shines alike on the palace and the dunghill."

Hilton's power of sarcasm, dreaded at all times, was on one occasion welcome. If any student had made himself obnoxious to his fellows, these comforted themselves with the thought that when he came under Hilton's hands, whether in the lecture theatre or the wards, retribution would be sure to follow. In the matter of doing prompt justice upon an offender, however, the palm must probably be given to Dr. Oldham.[1] It is said that a student, chiefly renowned for his want of manners, stood up during a midwifery lecture, and turned his back on the lecturer while he spoke to some friend in one of the rows above him. Dr. Oldham was more than equal to the occasion with "If there is one presentation which I detest above all others, it is *a back presentation.*"

For the following I am indebted to Dr. Savage, who, as is well known, was in the habit of giving Hilton back as good as he gave. "The first time I went round as one of his dressers I had a rose in my button-hole. Hilton merely looked at it. The next time I had a bunch of forget-me-nots. He looked, and said, 'Where did you get them?' I replied, 'From the country.' But the next occasion I had got a big thistle in my button-hole. To this Hilton said, 'Where did you get that? from the country too? I wish to goodness you would take half as much trouble about my cases as you do about your flowers.' I said, 'Tell me one of my cases

[1] Mr. Couling.

which is not properly dressed.' He laughed, and admitted that he had no ground for complaint.

"One of his dressers with a long beard asked permission to be absent for ten days. When pressed as to the reason, he owned that it was to go to St. Andrews for the M.D. After some hesitation Hilton said, 'Go, and I hope they will pluck you.' On the dresser's return Hilton said, 'Well, Beardy, did you get through?' 'Yes,' was the reply. 'And did they address you at the Northern Athens as *Hic ornatissimus vir?*'

"One day Hilton came round to find me alone of his dressers at the hospital, the rest being away at a cricket-match or something of the kind. 'Where are the rest of you?' said Hilton. I said, 'Not here.' 'I can see that,' he replied, and set to work to cut all the bandages, complaining of their not being well put on. He then told me to re-apply them before dinner-time. I said I would do as many as I could before, and the rest after. Hilton stormed, but I persisted that that was what I should do, and he became quite pleasant before he left. One Christmas-time, Hilton came round the day before. I happened to have 'taken in' a fortnight previously. He said, 'Where are you going to spend Christmas?' I replied, 'At home.' 'No, you are not,' he said. 'You are going to stop up and look after my cases.' I said I feared I should not; whereupon came the threat, 'I shall come round on Christmas Day and see if you are about.' I told him, 'Then you won't find me.' When Hilton came round next time he asked me if I had enjoyed the day, and was pleased that I had not been fool enough to mind what he said."

We have dwelt at some length on this element in Hilton's life, his brusque bluntness, his ready rebukes, too often sarcastic and irritating, and his too ready betrayal into frequently making jokes at other people's expense—jokes very often grossly personal, and on that account extremely galling. There is no defending or excusing this. Hilton himself, when he was dying, acknowledged this fault, when he felt that it was too late. It will suffice to remark that Hilton's early life, when a man is most impressionable for good or evil, had not been days likely to refine a man or make him

considerate of others, and out of these days Hilton had not come unscathed. Very strong in common sense, himself keenly observant and singularly accurate and ingenious in turning his observations into sound practice, he was unsparing at any want of common sense, and prompt to condemn any instance in which a dresser had lost an opportunity of observing for himself, and still more to ridicule any attempt to air a fad, or to quote some unproved and therefore unapproved theory. Too often, in thus condemning and ridiculing, Hilton went too far, and his condemnation and chaff passed into causticity, and his ridicule into rudeness.

But it is fair to add that though much has been heard of the rudeness and the ridicule, too little has been remembered of the approval and the praise which, in a few terse words, Hilton used to bestow on those who showed evidence of painstaking attention to details, shrewdness, and common sense—points which Hilton, being very strong in them himself, was always glad to recognise in others. We have seen, in Dr. Buck's words (p. lxx), that "strong men were powerfully attracted to Hilton as a teacher:" many of them can speak of instances of Hilton's roughness; but whether it is a prosector[1] who, falling asleep after a morning's hard work during the two o'clock lecture, was waked up with a rap on the head with a femur which lay too ready to Hilton's hand; whether it is an old dresser[2] who, giving an anæsthetic in private for Hilton and becoming too interested in the operation, received at the same time a smack on the face and the rebuke, "Just pay attention to the chloroform, and not to me,"—nearly all the victims (for there were many others) speak without bitterness of the stripes meted out to them; for they feel that Hilton's hand, if heavy, was impartial, and that his readiness with rebuke was intended for the student's good.

As an operator Hilton was most careful and attentive to details, but in no way distinguished for expertness or skill, or brilliancy in operating. To quote Dr. Buck again, "As an operator Hilton never played to the gallery. He was

[1] Dr. Constantine Holman.

[2] Mr. Couling.

painstaking and particular, finishing off all carefully, so that it was not often that his cases had to be taken down for secondary hæmorrhage, pain, &c." The bent of Hilton's mind, prone to watch and to observe, and to reason over his observations, was to delay resorting to the knife—a bent no doubt increased by the fact that he had begun his work in days before the introduction of anæsthetics. Hilton had seen that the great rapidity of work which was so necessary in those days had another, and, occasionally, a tragic side. He had, perhaps, been present, with the father of one of the most justly esteemed of the senior students now with us, when the deft hands of Aston Key, during lithotomy, an operation peculiarly his own, perforated the bladder.

Dr. Wilks, in his most interesting account of Key and Morgan, speaks of the brilliancy of the one and the skilfulness of the other as operators ('History of Guy's Hospital,' p. 337). Dr. Constantine Holman tells me that this rivalry was sometimes amusingly shown. Morgan and Key had each a case for lithotomy on one operating day. On Morgan's entering the theatre one of his dressers told him Mr. Key had just cut a boy in so many seconds. "Has he?" said Morgan, "d—d if I don't beat him;" and he did so on that day—by four seconds.

The very exactness of Hilton's anatomical knowledge helped to make him cautious, as is shown by the method of opening deep abscesses on which he laid such stress, and which still goes by his name. But that the same anatomical knowledge could make Hilton bold when boldness was required, is shown by his being one of the earliest surgeons to practise lumbar colotomy. Vol. xiii of these Reports (1867) contains a case of intestinal obstruction in which colotomy was performed, and twice repeated.

Nearly twenty years before, Hilton, while Assistant Surgeon, had, in January, 1848, operated on and reduced by abdominal section a case of obturator hernia which was simulating intestinal obstruction. The lady sank from acute peritonitis, and the specimen, a very valuable one, the relations of the parts having been dissected by Hilton himself, is in our Museum.

In 1846 Hilton had performed what he believed to

be the first recorded instance of any surgeon in this country having succeeded in his attempts to relieve an internal strangulation of the intestine by operation. This case was an extremely interesting one. The patient was a gentleman aged 21, whom Dr. Golding-Bird had seen in consultation, diagnosing strangulation of a loop of small intestine in the right iliac region, not far from the ileo-cæcal valve. This diagnosis was verified later when Hilton operated, and set free a loop of ileum, about six inches long, strangulated in an opening formed by a loop of small intestine, in its turn tied down to the brim of the pelvis by old adhesions, which probably dated from an attack of peritonitis originating "in mesenteric disease" when the patient was three years old. Here, too, the patient sank from acute peritonitis. When it is remembered that abdominal section was performed here without the aid of any anæsthetic—that, owing to the distension of the abdomen (the operation was performed on the fifteenth day of the illness), a prolonged search was required to find the strangulated portion of intestine—that in spite of all these difficulties Hilton succeeded in finding and in completely relieving the obstruction,—we, who operate with all the advantages of the close of the nineteenth century, will do well to remember Hilton's name with respect as one of the earliest pioneers in abdominal section for intestinal obstruction, under difficulties such as we shall never know.

Both these cases are recorded in the 'Medico-Chirurgical Transactions,' vols. xxx and xxxi. Each is illustrated with an excellent woodcut, Hilton having, *more suo*, made a necropsy himself in each case.

No account of Hilton would be complete without allusion to his Clinical Lectures. It was probably here that he was seen at his very best. In the first place he had an excellent report taken by a dresser, and to this report Hilton had given hours and hours of quiet thought and consideration. Only those who had to go to 10, New Broad Street, at what may be called odd times, realised how Hilton studied his cases.

It will be long before any teacher arises who has equal power of bringing out the importance of every detail in a

case, and of linking together, as he went along, a series of facts in the clinical report till they formed a continuous chain which kept the idlest student interested throughout the hour.

Happily a generation which knows not Hilton can still realise how he taught. Some of his clinical lectures will be found published in our Reports (vols. xi and xiii) for 1865 and 1867. A few were also published in the *Lancet*.

It is much to be desired that every Surgical Ward Clerk should read these Lectures before commencing his appointment. As we have stated above, Hilton always relied upon his dressers for the report of the case. When it is seen that one of these reports is drawn up by Mr. Howse, another by Mr. H. Morris, and that two of the series of lectures were arranged for publication by the Surgical Registrar of that day, Mr. G. Eastes, the excellence of the material which Hilton had ready to his hand to lecture upon will be recognised. The following well expresses Hilton's plan and object:—"I entertain the opinion that the best plan to pursue in a clinical lecture is to take up one case, to enter fully into all the details of the symptoms and treatment of it, and to explain to you to the best of my ability the why and the wherefore of everything associated with that single case. I consider my analytical plan to be productive of the greatest benefit to you, gentlemen, by inducing you to habitually examine and interpret every symptom presenting itself to your notice."

One of the most interesting lectures is the first in vol. xiii of these Reports, and headed "*Fatal case of rupture of left kidney, attended with hæmaturia. Remarks on clots of blood as aids to diagnosis.*" The man had been struck by the buffer of an engine in the abdomen, and, when he died on the twenty-sixth day, the left kidney was found to be ruptured. We will make a few extracts most characteristic of Hilton. "He breathed freely; in other words, he could, and did, use his diaphragm without inconvenience. . . The patient, however, could not cough. For this functional act the muscles of the abdominal parietes are used, and the patient's inability to cough was due to the fact that these

muscles were bruised. You must not ignore small points like these. Upon them the diagnosis of a case frequently depends."

"Later on, in the second day, the patient had inability to void his urine; why so? It might at first be from his position; for, when placed in bed on his back, a man often cannot easily pass his urine. Moreover the region in which the injury had been inflicted doubtless exercised much influence; for we find the bladder and urethra usually sympathise in various ways with kidney trouble, sympathetic spasm of the bladder being thus one of the most frequent concomitants of stone in the pelvis of the kidney. Moreover the abdominal muscles were partly paralysed from injury. Lastly, the bladder, recognising the fact of blood being in its interior, would telegraph to that effect to the urethral muscles, and the course of the urine would be arrested by spasm of the urethra."

"Up to the sixth day the case seemed to promise a good result. The blood in the urine was daily diminishing; the vomiting, pain, and other general symptoms were abating in severity. On the seventh day the patient had a craving for solid food; this was allowed him, and from this period he retrograded, his urine again became more bloody. The solid food was, of course, at once discontinued, but it had already induced some mischief. It necessarily produced an increase of the vermicular motion in the intestines, and also caused the heart to pulsate more forcibly; and now that we are familiar with the post-mortem evidence we can see how an increased force of the circulation might, and probably did, dislocate a clot which was closing the mouth of a torn artery. On the thirteenth day the patient was going on tolerably well, a little, though a diminished quantity of blood was still in the urine, but not enough to cause anxiety. On the evening of that day he vomited, and fresh hæmorrhage immediately supervened. I think that the recurrence of the bleeding was due to the vomiting, to the severe muscular efforts displacing a clot plugging a lacerated vessel. The pure blood was doubtless arterial, which traversed the urinary passages rapidly—so soon, in fact, that it had not time to coagulate in its transit."

Later on we come to a famous passage on hæmorrhage from the urinary tract:

"You know that extravasated blood, if it continues undisturbed in any cavity inside the body, will, in clotting, assume the configuration of that cavity. And so in hæmorrhage into the urethra; if the blood remains undisturbed there a little time, it forms clots which, being casts of the canal, are lengthened and cylindrical, or ovoid transversely. If there are several clots of cylindrical shape escaping in front of the urine, you may with certainty say that they are from the urethra. But if you want to ascertain exactly their form for the purpose of diagnosis, take a little trouble about the matter, and always swim out the clots in clear, cold water in a white dish or plate, giving them time to unravel themselves. If they are long and perfectly round, the site of their retention has been anterior to the prostate; if ovoid in their long axis, or like two triangles joined together at their bases, they were moulded in the prostatic portion of the urethra, and will probably be found grooved on their under surface. Fresh blood poured out rapidly will clot along the whole length of the urethra, like macaroni. But old clots from the urethra, numerous or single, may appear cylindrical, like pancakes rolled upon themselves, until swum out in water, when they perhaps unfold. This shows a slow and limited escape of blood into the urethra.

"With hæmorrhage into the bladder, the form of the clots is determined by the shape of the vesical cavity. When first passed from the urethra such coagula may seem to be cylindrical, so that you are perhaps inclined to say that they are urethral clots; but put them into a dish of water, they will then resume their flattened, circular, true shape. If the bladder is full of clot, nothing but blood-stained serum and urine will, perhaps, escape for days, until the clot is broken up by a catheter. Coagula formed within the bladder are somewhat irregular in outline, mostly circular flattened masses, bevelled off and fimbriated. If they have remained long *in situ* their fringed edges are white, being formed of decolorised fibrin. The fact that the shape of the clots in this case was not well defined was explained at the post-mortem examination; for the blood was not retained

at one part only, but, running from the renal vessels, collected in various spots of the ruptured pelvis and the ureter. Most surgeons throw away any clot that may happen to come from the invalid; but I would suggest to you never to do so until you have thoroughly examined it. Even if there is only one single confused mass, like that blocking the eye of the catheter in our present case, I advise you to swim it out in water; the shape that it then assumes may be of great help towards your diagnosis, for it will most likely exhibit the well-known form of the space that contained it during its coagulation.

"Take another outlet; let us pass to the consideration of hæmorrhage from the anus or rectum. A patient comes to you complaining that he is losing blood from the bowel. Your first question should be, 'Does the blood precede, accompany, or follow, or is it mixed with the motion?' If it comes before the motion, it probably arises from the lower part of the rectum. If it comes during the motion, it is probably caused by the feculent matter running across the plugged-up mouth of a wounded vessel and displacing the plug. If it comes after the motion, it is evidently associated with tumour, or something of the sort, higher up in the intestine. You should then ascertain whether the blood is fresh or clotted; and if the latter, whether the clots are new or old. If it is fresh fluid blood, or bright, arterial, and fluid, it comes from the membrane near the anus. The patient may inform you that something seems to protrude at defæcation; are you to be satisfied with this? No, it is your business to ascertain the source of the blood; as honest, legitimate surgeons, you must, for the benefit of your patient, take some trouble to determine the exact nature of the malady, however disagreeable the necessary proceedings may be to you. In the case I have cited you must send your patient to the water-closet, tell him to bear down thoroughly, and examine the protruded parts. In nine cases out of ten you will see at once the source of the bleeding." Hilton, continuing, goes on to propound the following:—"*A clot of blood may destroy life in various ways*, as after cut throat, when the external wound has been sutured before the hæmorrhage is arrested; in laryngotomy, tracheotomy, or

after the operation of harelip. *A clot of blood destined to arrest hæmorrhage may endanger life. A clot of blood may interfere with the success of a surgical operation.*" A wealth of illustrative cases, all eminently *ad rem*, is quoted to illustrate these maxims.

Another excellent lecture is one on tetanus. It is headed "*Simple fracture of femur. Extensive laceration of the integument. Tetanus. Recovery.*"

"Each case of fracture must be competed with on its own merits, with that kind of splint which retains the fragments in a position the nearest to the state of health. I want you clearly to recognise why in one case you employ this, and why in another instance you use that, apparatus. In this, as in all cases, pray reason for yourselves, and do not accept, without scrutiny, the dicta of the books.

"On the third day the tissues of the upper part of the thigh, which were bruised and in fact detached at the time of the accident, had become sphacelated; and the site of mortification was over the line of the femoral vessels. It was at first impossible to determine how deeply the gangrene might extend, and therefore the dresser was requested to be upon the alert, in order to compete with any hæmorrhage that might occur on the separation of the slough. But, providentially, no large vessel was opened. You may hereafter be called to attend to a large wound over the femoral vessels, and you will then be anxious to know whether hæmorrhage is likely to ensue—in other words, whether the artery is injured. If you find no interference with the temperature of the foot, and no interference with its arterial circulation, you have two points that favour greatly the idea that there is no injury of the femoral artery. But if, with a deep wound of the thigh, the foot is losing its temperature, or the pulsation in the tibials is much diminished, you must be on the look-out for secondary hæmorrhage, and be ready to adopt immediate measures for its arrest. With a large granulating surface over blood-vessels you may fairly still entertain a slight apprehension of hæmorrhage. If, however, the granulations are healthy, the underlying tissues are healthy also, the sheath over the femoral or other vessels

(as the case may be) will probably not give way, and you may dismiss all fear of bleeding.

"The first indications of tetanus, which occurred on the thirteenth day, were 'jumping' of the fractured limb and rigidity of the lower jaw. The 'jumping,' as here displayed, has its origin in the central part of the spinal marrow; in other words, is due to some local disturbance of the grey matter. Trismus signifies something amiss with the fifth nerve, which supplies all the muscles of mastication. I cannot tell you exactly why this should be so, why those muscles should be almost invariably the first in which tetanic symptoms are manifested. But I think we may find an explanation in the fact that the sensitive part of the fifth nerve has very extensive structural connections with the grey matter of the upper part of the spinal marrow, and that, having more association with this same grey matter than any other nerve of the human body, the two necessarily sympathise to a very great degree.

"The pulse and respirations were scarcely at all quickened. I wish you to notice these points particularly, and also the fact that there was no difficulty in swallowing. What do you, as anatomists, infer from these statements? The pulse not being above 88 one day, nor over 94 in the next, nor above 100 at any time, showed that the heart was not very irritable, and that the ganglionic nervous system was not seriously implicated. Further, the pneumogastric nerves were not affected, since respiration was but slightly quickened, and deglutition not difficult. The boy could swallow perfectly well, consequently his pharyngeal muscles were not involved. His voice was clear, and not at all changed in character; this showed that there was no spasm of the larynx.

"It may be distressing for you to witness the violent spasms which affect the patient's voluntary muscles, but you must at the same time remember that he will not die from those. His danger must be measured by the difficulty of respiration and of deglutition that he experiences, and by the frequency of the cardiac pulsations. The chief peril arises from spasm of the heart or of the laryngeal muscles. I wish you, then, to look into a case of tetanus with care and precision, especially in regard to prognosis, and not to dis-

miss every case as hopeless. It is a good old professional saying that if the patient can swallow and breathe freely he will recover. Experience also teaches that if the pulse remains below 90 or 100, and the respirations are scarcely if at all quickened—that if, in other terms, neither the pneumogastrics nor the ganglionic nerves are implicated—the prognosis is good, but that as soon as the heart becomes importantly involved the prospect is almost hopeless."

The following paragraph shows—oddly as the treatment reads now-a-days—how near Hilton, with his keen powers of observation and sagacity in reasoning, had come to the great principles which lie at the root of Sir J. Lister's discovery.

"The wound in the thigh was dressed with Carron oil to prevent the escape of heat from the part, for there was a tendency to gangrene. Besides, I had another object in view, for which I ordered the dressings to be changed every third day only. This was to exclude the air, which acted as a local irritant, and was prejudicial to the boy in his then tetanic condition. The wound beneath the lint and Carron oil became covered with thick creamy pus, by which the access of air was prevented. But some one will, perhaps, be inclined to ask, 'Is atmospheric oxygen an irritant?' I will cite a few instances in which it certainly seems to be so. I have already referred to cases in which there is a very painful fissure or 'hangnail' near the root of one of the finger-nails, which, after resisting all ordinary treatment, may be cured in two or three days by placing a small piece of plaster over it. So, again, if the whole surface of a long-standing painful ulcer be touched with nitrate of silver (by which a covering pellicle of coagulated albumen is formed) the ulcer beneath will often be quickly repaired. Lastly, dust a thick layer of flour upon a portion of skin attacked with erysipelas, and note how quickly the pain and irritation will subside. Now the common source of benefit in all these instances seems to me to lie in the exclusion of the atmosphere from the diseased surfaces."

The most important of the other lectures are on fractured clavicle, senile gangrene, strangulated hernia, irreducible hernia, fractured spine, injury to the head, and concussion.

With two more abstracts we will close this reference to Mr. Hilton's clinical lectures. He is speaking of the case of injury to the head :—"This boy was suffering, on admission, from concussion of the brain, and not from compression. Even though there apparently be unconsciousness in concussion, still you then have a power of arousing the brain, as was done in this boy's case; but you cannot so influence the brain, which has suffered sufficient structural injury, as to cause symptoms of compression. In arousing the patient, do not rest satisfied with simply asking the patient how he feels, or how his accident arose. If you merely speak to him, calling him by his name, he will not answer you; but if you pinch and pull his nose, and he has within even the minimum of consciousness, he will recognise that act as an indignity, and will endeavour to resent the insult, or will flinch or turn himself in bed. Moreover, learn from some of his friends the Christian name by which he is usually accosted; and then shout in his ear your question, addressing him first of all by the term most familiar to him, as Jack, Bob, Bill, or whatever it may be. If conscious, he will then reply to your queries."

Our last extract is from a clinical lecture on a case of "*irreducible inguinal omental hernia*."

It is a good instance of how Hilton made his early anatomical training subservient to surgery, and his ingenious way of reasoning from anatomical facts. It proves how habitually he made matter-of-fact details interesting—to many minds, we might say, fascinating; and it recalls those lectures where all listened intently while the surgeon spoke, and all who listened learned.

"Where the patient is always walking about, an irreducible hernia is a great nuisance, and may induce, occasionally, strangulated bowel. I have had several cases come under my notice where the patients were willing to put themselves under surgical control for a month or so, provided they could be quite sure they would get rid of the rupture. Here, then, comes an important question,—How are you to reduce an irreducible hernia? You must make the patient lie continuously on his back; lift up the scrotum, apply ice in a bag continually to the tumour; give him Hyd. c. Cret.,

or some mercurial, each night, and a dose of white mixture (Mist. Magn. c̄ Magn. Sulph.) in the morning. Let him take good meat diet, and as little fluid as possible. Why all this? We must remember that the greater part of each of you is fluid, and that the walls and contents of the hernial sac are no exception to this general rule. Now, by making the patient lie continually on his back, and by elevating his scrotum, you facilitate the flow of venous blood from the hernia, and consequently take off a deal of congestion to which the ruptured parts were subjected so long as the patient stood or sat up. But you must impress upon him the fact that, however disagreeable to himself, he must nevertheless keep always on his back. Should his bowels be relieved, he must have a bed-pan placed beneath him. Inflexibly, I say, you must make the patient lie continually on his back in bed, with his scrotum well up. He must not be allowed to get up on any account whatever. You will, perhaps, lay great stress upon this point when you remember that the veins of the omentum have no valves—that they have no attendant arteries from the pulsation of which the venous blood-current can receive impulses to its onward course; and they, further, have no muscular force like that in the midst of which lie the veins of the extremities to aid in the accomplishment of the circulation. Why is the ice used? Cold lessens the afflux of arterial blood to a part. When applied to the tumour it diminishes the flow of blood through the vessels of the walls of the sac, and influences similarly the quantity of the blood circulating in the capillaries of the omentum or intestine. It may, in fact, do this too much, and cause gangrene. A few years since I saw a case of large umbilical hernia, with a thin layer of integuments over it, and in an old person, to which ice was applied for a few hours only; yet upon operating I found the intestines pale, flaccid, cold, stinking, and gangrenous, owing to the deprivation of blood by the too intense cold which had been employed. If the person be old and weak, and you judge him not capable of withstanding much continued deprivation of temperature, you should take away the ice and apply an evaporating spirit lotion, which is not so cold. Why use the mercury and chalk?

The contents of the sac are often glued together and to the surrounding walls by recent or old adhesions. Now mercury deteriorates the lowly vitalised fibrinous matter of which these adhesions are composed, and then the healthy serous membrane absorbs it. If irritated, serous membranes are secreting sacs, but they become absorbing surfaces as soon as the secreted materials forming the adhesions are deteriorated. *Rest* reduces notably the thickening produced by old inflammation of joints; similarly you may hope to aid absorption of the hernial adhesions by removing all disturbance of the parts, which is best done by keeping the patient quiet in bed. Why give M. M. c̄. M. S. each morning? I believe you can unload the portal system to a great extent by Epsom salts. Give also as little fluid nourishment as possible, for fluid occupies a considerable space. Meat is the best food on this account, and all apprehension of constipation is avoided by the daily dose of the white mixture."

Before leaving Hilton as a Clinical Teacher, we must refer to his *magnum opus*, his book on Rest and Pain, more exactly called 'A Course of Lectures on the Influence of Mechanical and Physiological Rest in the Treatment of Accidents and Surgical Diseases, and the Diagnostic Value of Pain.' These lectures were delivered at the College of Surgeons in the years 1860, 1861, and 1862. But of this book, one of our very few surgical classics, we shall prefer to quote the words of others. A reviewer in the 'British Medical Journal,' in noticing the second edition, which appeared in 1877, said, "Mr. Hilton's lectures on Rest and Pain are, perhaps, the most considerable contribution to surgical literature furnished by any of the professors of surgery who have occupied the chair at the College of Surgeons since Sir James Paget's celebrated lectures on 'Inflammation and the Healing Process and on Tumours' were delivered." The same reviewer pointed out as the chief merits of Mr. Hilton's book that it rebuked, and provided an admirable corrective to meddlesome and hasty surgery. Again, it abounds in the ingenious application of the facts of ordinary dissecting-room anatomy to the requirements of daily practice. "In this volume we have indubitable proofs that a

patient may have suffered for years from symptoms which might have been relieved by the simplest means if the distribution of the nerves had been known and borne in mind, but which, for the want of such knowledge, or of its application, were attributed to all sorts of causes erroneously, and therefore treated with entire want of success."

A little later in the review the following passages occur:

"Can anything be more exquisitely ingenious? All that the severest critic could say is that it is, perhaps, too ingenious to be true; yet, true or no, what a light does it throw on anatomy! what an interest does this way of speculating upon facts give to their investigation! Even the errors of such a master may be rich in instruction."

"We have made these few observations on Mr. Hilton's lectures, not because we fear that any of his former hearers or readers can have forgotten his teaching, but in order to kindle a younger generation to study one of the most valuable, most practical, and at the same time most ingeniously speculative of all surgical works. Life moves fast in these railway times, and one of the evils of our rapid movement is that we are very apt to be content with a hurried glance in passing, and speedily forget things which deserve patient study and long familiarity. Such is Mr. Hilton's work; one that the student should use as his 'enchiridion,' till he has not only become familiar with the matter of his author, but has imbibed his author's habit of mind and way of study, and has prepared himself to investigate the bewildering phenomena of disease, mental and physical, with the same reliance on physiological science, the same patient observation, and the same fertility of resource."

Much more lately, in August, 1891, nearly thirteen years after Hilton had been laid in his grave, another voice was raised in praise of the author and his book. Prof. Chiene, when giving the Address on Surgery at the meeting of the British Medical Association at Bournemouth, took as his subject "Rest as a therapeutic agent in surgery." The following pithy quotation is some of the highest praise that Hilton has ever had:—"Bacon says there are books to skim over, books to read parts of, books to absorb; Hilton's book on 'Rest and Pain' is one to absorb."

This praise would have been especially gratifying to Hilton, for it is an open secret that the living surgeon who thus reverenced and praised the dead is known at Edinburgh as "honest John."

Hilton resigned his full surgeoncy in 1871, after a tenure of two-and-twenty years. As we have written this account for Guy's men, to recall him to the memory of those who honoured and valued him, and to enable generations who knew him not to form a better idea of the man, as he worked for forty-three years at Guy's, our space does not admit of our following his life beyond the walls of our hospital.

It remains to try and sketch the appearance of the man. The photograph at the beginning of this article was the only one available; it was taken after Hilton's prime, and before old age (which never came to Hilton) had had an opportunity of softening the less attractive features of his face. It does[1] Hilton justice in one sense only, and that is—"justice without mercy."[2]

No one would have thought, in looking at Hilton, that he had a great Surgeon before him. He looked much more like a prosperous City man. Short, rather stout and dapper, in a plain, black frock-coat, with faultless shirt-front and linen, a black stock or bow-tie, a fancy waistcoat, festooned with one of the old-fashioned gold chains, very neat, however, and boots with irreproachable blacking—such was Hilton's outer man as he got out of his carriage to enter the colonnade, or as he sat upon one of the beds in his wards examining an inflamed ulcer with a probe, that he might detect the position of the nerves which were exposed in the ulcer.[3]

[1] To alter slightly the old saying, "lenit albescens *faciem* capillus."

[2] I think it is the author of the 'Horæ Subsecivæ' who says that photographs are rarely first-rate unless they be of dogs or children, or of old people, whose faces, like other old houses, are often picturesque. It is certainly that most genial and gentle of men who wrote, "Richmond pleasantly put it, and truly, to Professor Syme, when he first showed him the drawing of himself well known by Holl's engraving; the great surgeon, after scrutinising it with his keen and honest eyes, exclaimed laughing, 'Yes, it is like; but then—it is good-looking!' 'Ah, you see,' said the artist, 'we do it *lovingly*.' The best likeness of a man should be the ideal of him realised." The difference between "a Richmond" and a photograph could not be better put.

[3] To quote Dr. Wilks, "it might be a simple ulcer, which he would dilate upon, until the student beheld in it something much more worthy of note than he had

Any notice of Hilton, however incomplete, should refer to the fact that certain honours fell to him, unsought, which rarely come, together, to any one Surgeon. He was appointed Surgeon Extraordinary to the Queen. In 1871 the Pathological Society, mindful of Hilton's services in years long gone by in the advancement of sound Pathology, did Hilton and itself honour by electing him President—a compliment especially high, as Hilton, though an original member, had never contributed to the 'Transactions' of the Society. But the highest of these honours was the first; in January, 1839, Hilton was elected a Fellow of the Royal Society. In the paper by which he was recommended as a Fellow he was described as "a distinguished anatomist and lecturer on descriptive anatomy at Guy's Hospital; a gentleman much attached to the sciences of human anatomy and physiology."[1] The actual work specified in the above paper as done by Hilton was, of course, the two anatomical papers published by him in our 'Reports' for 1837, one on the "Distribution and Probable Function of the Laryngeal Nerves," the other on the "Sacculus or Pouch in the Human Larynx." Hilton describes this pouch, its position, structure, and capacity, together with the numerous glands supplying it, and the muscles which act upon it; he then discusses the offices which it performs, connected directly and indirectly with the functions of the larynx. In 1838 Hilton contributed a paper to the 'Proceedings' of the Royal Society, on "The Decussation of the Fibres at the Junction of the Medulla spinalis and Medulla oblongata." It is superfluous to add that the only books which he published afterwards, viz. his 'Lectures on the Cranium' and his 'Rest and Pain,' were entirely worthy of the honour which the Society had done him, very early in his career, when only thirty-one.

The closing years of Hilton's life were overclouded with

first considered it. It was a case of this kind in which he first discerned that the ulcerative process was kept up by the irritation of a nerve, upon the division of which the sore healed. The case became classic, and the treatment was looked upon as a great discovery, for it was long before neuritis and the effects of nerves upon nutrition had been carefully studied, and the knowledge thereby gained became the property of the profession. Other trivial complaints Hilton treated by the same scientific method.

[1] Dr. Pye-Smith.

increasing distress and suffering, owing to a carcinoma of the stomach, which proved fatal in September, 1878. But those who were much with Hilton at this time noticed that, as he faded, he softened. Many of the characteristics of old days were present almost to the last—the sagacity, the honesty, and the pungency ; but the sharpness, the austerity, perhaps we should add the bitterness, were largely gone.

One matter should be mentioned as bearing on what we have said about Hilton's blunt brusqueness, his ready rebukes (too often sarcastic and irritating), and his too easy betrayal into making jokes at other people's expense. He was on his way to Eastbourne—from which he returned quickly, and but a few days before his death—and met Mr. Couling of Brighton. After referring to his illness and fading strength, Hilton said he had been looking backwards, and added, "I know I used to be down upon the men, but I did it for their good." His old dresser and house-surgeon —then, as ever, prompt to cheer and soothe, and to say the kind word—comforted Hilton with the assurance, "I don't believe there is an old dresser of yours that doesn't think of you at least once in the day." "Do you think so," replied the dying man, "upon your honour?"

And now my task is ended. Meagre, unsatisfactory, and far short of my desire as this article is, I trust I have enabled those who knew not John Hilton to think of him as a truly great, because a physiological and scientific Surgeon, and, as a man, as honest and straight as a man can be. My one endeavour has been with a grateful hand to trace, as faithfully as I know how, those personal outlines, and to fix those vanishing traits, which will enable posterity to identify and to individualise the man. A few years ago these dwelt very vividly in the minds of not a few of us, far better qualified than myself to do honour to Hilton's memory. Pass a few more short years, and these outlines, these traits, will have died out of men's remembrance, and, once departed, such things can never be recalled. Whether it is a student in his earlier years, when working at the anatomical models, or one of us of a senior standing, who profit by those teachings by which John Hilton contributed so largely to the more scientific treatment of surgical diseases, we should,

one and all at Guy's, honour his name with the words on Wolsey's bust at Christ Church:

NOMEN INTRA HAS ÆDES SEMPER VENERANDUM

W. H. A. Jacobson.

It is very pleasing to relate that our School has lately received £100, to be spent in honouring and perpetuating Hilton's name. The donor is a warm admirer of Hilton, and though not a Guy's man, has sent two sons to us.

Yours ever sincerely
Edward Lowe

# A BIOGRAPHICAL SKETCH

OF

# EDWARD COCK, J.P., F.R.C.S.

By R. CLEMENT LUCAS, B.S., F.R.C.S.

The lives of great institutions are permanent relative to the workers who toil therein, and to a few only is it given to engrave their names indelibly upon the walls. Among these some there are who, fired with burning genius, rise rapidly, and, like the brilliant meteor's golden course, as quickly disappear, leaving us for a time in saddened darkness; others, again, like the subject of this memoir, gifted with sound constitutions and well-balanced intellects, toil on whilst years upon years roll by. To these, who like the planet's even glow work steadily on unscathed by time,—to such as these, the establishment owes its greater debt, since their great names act as more stable buttresses to the support of its permanent reputation. For more than half a century has the name of Edward Cock been associated with Guy's Hospital, wreathed with its best traditions, and bound up in many a humorous story, of which he probably was often as remote an author as Horace or Juvenal; for to a wit of established humour all new witticisms are liable to be attributed by those who dare not father their own offspring, and in the medical world perhaps no one has suffered more than Mr. Cock from

this affiliation of bastard stories. Still, his genial bachelor ways, his openly avowed dislike of all characters of the Squeers or Stiggins type, his tolerance and forgiveness of youthful indiscretion, and, above all, his fascinating stammer, combined to individualise him as a man, on whom to father a story was to double its point and humour.

Many, indeed, are the tales which in their rooms of an evening students loved to recount of their kind-hearted and genial teacher, and as each story by repetition grew, their wonders increased, till the morning mists dispelled the midnight cloud, and the thoughts of early anatomical lecture warned the convivial group of the necessity of retiring to rest. For to the student the most interesting topic of conversation is ever the relative worth of, and the little peculiarities which distinguish, their respective teachers; but no one's mental bent ever afforded so fruitful a theme for discussion, as did the distinctive traits in the many-sided character of their much beloved lecturer, Edward Cock. It was his boyish and sympathetic heart, which remained unsoured and unsuppressed by age, that kept him quite to the last in touch with the effervescing fun of youth.

Edward Cock was the third son of Mr. John Cock, an underwriter at Lloyd's, by his marriage with Maria, daughter of Baron Hesse, of Potsdam, in Prussia, and was born at Tottenham, Middlesex, on January 26th, 1805. He used to say that he owed most of his education to his mother, whose knowledge of Continental languages was imparted to him in early life, and enabled him, when commencing his professional career, to gain some means of livelihood by translating foreign papers for the medical periodicals. His education in modern languages was further encouraged at the school of Dr. Schwabe, a German, of Stamford Hill, where he was afterwards placed. Two generations back the family of Mr. Cock was connected with that of Mr. Cline, the distinguished surgeon of St. Thomas's Hospital, to whom Sir Astley Cooper was articled, and it was through Mr. Cline's introduction that Sir Astley met his wife at Tottenham. Lady Cooper was the daughter of Mr. John Cock, a wealthy merchant, whom Cline used to visit on Sundays, and she was Mr. Edward Cock's aunt. Being in this way closely connected with the greatest

surgeon of the day, young Edward Cock's thoughts were soon directed towards the medical profession, and we find him at the early age of sixteen apprenticed to his distinguished uncle, Sir Astley Cooper, and commencing his studies at St. Thomas's Hospital, where, as the two Borough Hospitals were then united, the surgical and anatomical part of the school was carried on.

Mr. Cock's fellow-students have all too long passed away to allow of my obtaining any direct evidence of his student days; but that he worked diligently we have ample evidence from the position he afterwards obtained, yet to suppose he was of the cadaverous ascetic type that sneers at healthy enjoyment would be to belie all that his after-career renders probable. Far more likely is it, that the nephew was but little behind in the boyish freaks and practical jokes for which the uncle's youth was chiefly distinguished, and that this tendency to fun and genial companionship, which they evinced in common, had something to do with the depth of friendship which sprang up between the teacher and pupil. Certain it is, that Mr. Cock's admiration of his illustrious teacher developed into a hero-worship so strong, that in his desire to give all credit to his uncle, one felt he often did scant justice to himself. To use his own words, "the warmest feelings of mutual esteem and friendship always existed between the illustrious master and the pupil, which only ceased with the death of the former."

Sir Astley, in his earlier days, was a frequent visitor both at Mr. Cock's grandfather's and at his father's house. The grandfather died on November 21st, 1791, the very day that had been fixed for Astley Cooper's wedding to Mr. Cock's aunt; in consequence of which the wedding had to be for a time deferred. Through the kind offices of Mr. Cline, however, this wedding, which was to exert so great an influence on Mr. Cock's life, was not long deferred, though out of respect for the dead it was considered right that it should be strictly private. In the following month a Christening was to take place from Mr. Cline's house, and this good friend invited the young couple to accompany the Christening party to the church, where the marriage was solemnized, and they afterwards retired as if they had been merely witnesses of the

baptism. The secret was so well kept that the young surgeon met his class in the theatre the same evening, and delivered his surgical lecture with perfect ease, as if nothing unusual had happened, and afterwards went to the house in Jefferies Square, which had been furnished for him by his deceased father-in-law.

Before Edward Cock had finished his apprenticeship, in the commencement of the year 1825, began those stirring times which led to the separation of Guy's from St. Thomas's Hospital, and the foundation of the Guy's Hospital Medical School as a distinct establishment. At this time Astley Cooper was in his fifty-seventh year and in the zenith of his fame, a baronet, surgeon to George IV, and senior surgeon to Guy's Hospital. In the discharge of his many duties he felt the lectureship on surgery, which was held in St. Thomas's Hospital, pressing too heavily upon him, and he determined to send in his resignation. For some time previously, his nephew, Mr. Aston Key, had been delivering part of the surgical lectures, whilst another nephew, Mr. Bransby Cooper, had given the lectures on anatomy. It was usual in those days for the retiring lecturer to nominate his successors. No sooner, however, was Sir Astley's resignation received, than the Treasurer of St. Thomas's Hospital appointed Mr. South to the Lectureship on Anatomy in the place of Mr. Bransby Cooper. It was fortunate for the future fame of Guy's Hospital that the Treasurer of that day was a man of singular ability and great resolution. He sympathised with Sir A. Cooper, and expressed his indignation at the slight which had been cast upon him, then proposed to found a separate School of Medicine at Guy's Hospital, and to appoint Mr. Aston Key and Mr. Bransby Cooper to the respective chairs of Surgery and Anatomy. This was at once agreed to, and steps were immediately taken to carry the idea into effect. Thus Sir Astley Cooper and Mr. Benjamin Harrison, the Treasurer, may be regarded as the founders of the Guy's Hospital Medical School.

The Treasurer was loyally supported by the Governors in carrying out the idea of a separate school, and we find him authorised "to make such arrangements as he may find necessary to provide for, and place the surgical school of this hos-

pital, in all its departments, on the most respectable and efficient footing." With such extraordinary promptitude and energy was the scheme carried out, that, although the idea was only broached at the end of January, by the following October the present anatomical theatre and museum, including a dissecting-room, were completed at a cost of £8000, and ready for occupation. Three of Astley Cooper's nephews found positions in the new school, Aston Key took the chair of surgery, Bransby Cooper that on Anatomy, and Edward Cock, though only twenty years of age, and with his apprenticeship not yet completed, was appointed Demonstrator of Anatomy in the dissecting room. It was not to be expected that such an apparent family arrangement would pass without criticism; but the world-wide reputation and great popularity of Astley Cooper overwhelmed all opposition. The students flocked to Guy's, and left St. Thomas's almost deserted. Bransby Cooper was selected to give the introductory lecture; but here a stormy scene occurred, for the opposition appeared in force. Up jumped Dr. Cholmondeley, a physician to Guy's Hospital of aristocratic connections, and delivered himself of a violent diatribe, imputing interested motives to Astley Cooper. Dr. Cholmondeley's character is perhaps best illustrated by the story I heard from the lips of the late Mr. James Stocker, that when the stethoscope was first introduced, he appeared with one in his button-hole, and a rose fixed in the end of it, to show his contempt for the new invention. The *fracas* was reported to Astley Cooper, who sent a challenge to Dr. Cholmondeley to publicly apologise or fight a duel. The following are his own words relating to the event:—"Dr. Cholmondeley, of Guy's Hospital, had the impudence to charge me with acting interestedly in this affair. As soon as I heard of the charge against me, I had no hesitation in making him retract his words or meet me. I therefore sent Mr. Morgan to him, to tell him he must publicly withdraw his accusation in the theatre of the hospital or meet me hostilely, and he fixed upon a Mr. Power to do as he thought right for him. Mr. Power said he thought Dr. Cholmondeley had been wrong, and ought to publicly retract, which he did to Mr. Morgan's satisfaction. Bransby read his recantation in the theatre, and thus a duel was prevented."

Next arose an angry discussion over Sir Astley Cooper's

pathological specimens, which had been deposited in the museum of St. Thomas's, and which he desired to recover. These, however, the Treasurer of St. Thomas's refused to deliver up. The new staff of Guy's were, therefore, stimulated to use every effort to fill the vacant shelves, and we find Edward Cock taking an active part in the arrangements of the new school, and exerting himself especially in the formation of an anatomical museum. From this time forward, during the twenty succeeding years, he continued to teach anatomy in association with Mr. Hilton, and only resigned when the duties of Hospital Surgeon, and his increasing private practice, added to the wear and tear of lecturing, began seriously to undermine his health. On his retirement from the anatomical chair the former pupils of the hospital, who had studied anatomy under his tuition, availed themselves of the opportunity to present him with a handsome piece of plate.

Mr. Cock became a member of the Royal College of Surgeons in 1828, and an Honorary Fellow of the first twenty-seven created in 1843. It was not, however, until he had spent thirteen years in the dissecting-room that he was appointed Assistant Surgeon to Guy's Hospital; but for some years previously the engagements of Mr. Key and the absence of Mr. Morgan, who lived out of town, had thrown upon him the active duties of hospital practice, and had afforded him the frequent opportunity of performing many of the major operations of surgery. Thus he served a long probationary period, and had shown himself well qualified for the post, when, in 1838, he received the appointment. It may be said that Cock owed his demonstratorship in the dissecting-room to his uncle's influence; but that influence had long passed away before he received his appointment on the permanent hospital staff. He was then standing on his own merits, and his great popularity with the students must have assured his success. He had escaped from the shadow of his relative's name and stood alone in broad daylight. For my part I much prefer a direct to reflected light. There is a clearer outline and a deeper shadow. Many, however, owe their whole distinction to the reflected greatness of others. It cannot be said that any one of the three nephews, whom

Astley Cooper introduced, failed to justify his selection or threw discredit on his judgment. Cock outlived the other two by over forty years. He never lost his gratitude for his first assistance, and never did anything that could throw the slightest shade of disgrace on his uncle's name; but keeping his mind open for new facts in a most remarkable way to the very last, he used his experience for the assistance of his juniors, and for the benefit of the institution to which he was attached.

During the period he was employed in teaching anatomy, Mr. Cock made many dissections of the vessels and muscles, which were dried and varnished, and are still preserved in the museum. In the first volume of the 'Guy's Hospital Reports,' published in 1836, we find a description of two elaborate dissections, illustrated by admirable coloured plates, of the collateral circulation after ligature of the main vessel of the limb. One of these is a case of ligature of the external iliac artery by Astley Cooper, which Cock had an opportunity of dissecting eighteen years after. The other is a case of ligature of the subclavian for axillary aneurism by Mr. Key, which Cock dissected twelve years after the operation. These cases have become historical; but the credit of the dissections has been hidden by the fame of the operators, Mr. Cock's name being only mentioned in the text; yet, so far as the anatomical interest is concerned, the credit should rest entirely with the dissector.

In the second volume of the 'Guy's Hospital Reports' we find Sir Astley Cooper has been stimulating his young anatomists to work out the origin and distribution of the superior laryngeal nerve. Mr. Cock examines its origin and relation to the lower ganglion of the pneumogastric, which Astley Cooper had discovered, whilst Hilton works at the distribution of the branches. The opening sentences of these papers are so characteristic of the two men, that I think it will be of interest to quote them. Cock begins:—"I have been induced to draw up the details of this paper in consequence of a communication received a short time ago from Sir Astley Cooper. It will be seen that I have attempted nothing more than to follow out his views on the subject, and to enlarge somewhat on a discovery which is exclusively

his own." Hilton commences as follows :—"Considering the following description of the two laryngeal nerves not merely a record of my opinion, but a statement of facts acquired from repeated and careful dissections of my own, I do not deem it essential to its interest or importance that I should attempt to expose the probable causes of the various discrepant opinions already published in reference to their distribution." Both papers show excellent work. Cock describes the form of the ganglion and its relation to the superior laryngeal nerve in the horse, ass, sheep, dog, cat, rabbit, rat, ferret, guinea-pig, and man; whilst Hilton gives an elaborate account of the terminal filaments of both laryngeal nerves in the human subject; and each author refers to the other's paper, showing that they must have worked together.

When demonstrator of anatomy, Mr. Cock published a book entitled a 'Dissection of the Vessels and Nerves of the Head, Neck, and Chest,' which was familiarly known as "Cock's Head and Neck." This work, though incomplete as not embracing the whole body, was remarkable in the originality of its design, for it was the first treatise in which the different parts were described as they are successively brought into view under the scalpel of the dissector. The plan was afterwards copied and more elaborately carried out by Ellis in his 'Demonstrations on Anatomy.'

In the 'Medico-Chirurgical Transactions' of 1835 there is an original and excellent paper by Edward Cock, Demonstrator of Anatomy at Guy's Hospital, on malformations of the internal ears as a cause of congenital deafness. In this paper he describes the pathological changes which he discovered in five patients who died in the Deaf and Dumb Asylum, an institution to which he afterwards became attached as consulting surgeon. All the cases had died of strumous disease of the thoracic or abdominal organs, and three of them he rejects as having recent tubercular disease of the ear. "I merely mention these facts," he says, "as indicating the strumous habit of body, which I believe prevails very generally among the deaf and dumb." "I may also remark, that in all the cases examined, the petrous portions of the temporal bones exhibited more than the usual varieties in size and shape. In some the bone was so deficient

in particular spots as barely to cover the internal cavities, whilst in others there appeared a preternatural osseous development." The pith of the paper, however, rests in the discovery, in two instances, that two out of the three semicircular canals of the bony labyrinth in their central portion "were impervious, or rather did not exist at all." In one ear also the fenestra rotunda was found closed by bone. Here then is a definite pathological discovery, worthy to be ranked with Hilton's discovery of a closed aqueduct of Sylvius in association with hydrocephalus. The paper is illustrated with woodcuts, showing the extent to which the canals were defective, and it shows too that the author was familiar with the comparative anatomy of the organs on which he was writing. A sixth case was added after the paper was in the press. In this he found the fenestra rotunda closed by bone on both sides. His explanation of the result of such a defect is as follows :—"If we consider the labyrinth of the ear as a long osseous tube, commencing at the fenestra ovalis and terminating at the fenestra rotunda (and thus closed at both extremities by membranes), then if a solid structure be substituted for the yielding material, which, in the natural state, closes one extremity of the canal, viz. the round opening, the motion imparted by the ossicular to the membrane of the fenestra ovalis would no longer produce that undulation of the fluid through the labyrinth, which appears essential to the appreciation of sound." Only those who know the difficulty of extracting the temporal bones from the skull, and the still greater difficulty of dissecting its petrous portion, can appreciate the labour which resulted in the discoveries enumerated. Before Cock's observations, congenital deafness was "almost universally ascribed to paralysis of the auditory nerve."

The volume of the 'Medico-Chirurgical Transactions,' in which this paper appears, is an interesting one, for it contains the Royal Charter granted in 1834 to the Society as the result of a petition signed by John Elliotson, Doctor of Physic, Sir Astley Paston Cooper, Baronet, and John Yelloly, Doctor of Physic. It is easy to see who of the three was the prime mover in obtaining the recognition of Royalty for this Society, which was formed in 1805, with the Guy's Physician, Dr. William Saunders, as its first President.

Three years later, in the 'Guy's Hospital Reports' for 1838, Mr. Cock having had opportunities of dissecting the ears of seven other patients who had died in the Deaf and Dumb Asylum, contributes another and fuller account of the conditions discovered. He thus sums them up :—(1) Adventitious growth filling up the tympanum. (2) Deficiency of the fenestra rotunda. (3) Partial or complete deficiency of the spiral canals of the cochlea. (4) Preternatural enlargement of the aquæductus vestibuli. (5) Deficiency of the semicircular canals. (6) Unnatural solidity of the temporal bones. The paper ends with one of those eloquent paragraphs for which his papers are always distinguished—"Although," he writes, "I cannot flatter myself that the issue of my labours has afforded any information that can be attended with any beneficial result in a practical point of view; although the establishment of a fact, which has been denied, that congenital malformation may and frequently does exist, would rather discourage us from adopting remedial measures, than serve to indicate any mode of treatment for the relief of the patient; yet I am not without hope that my investigations may still prove of some value in the hands of those who undertake the treatment of deafness on scientific principles, and with a view to the benefit of their patients as well as their own emolument. I trust they may stir up a spirit of enquiry into the symptoms and degrees of deafness, which may lead to the establishment of a more accurate and efficient diagnosis, and enable the aurist to distinguish between those cases which are irremediable by art, and those which may be expected to derive benefit from judicious treatment."

In the 'Guy's Hospital Reports' for 1841 is a contribution on "Stricture of the Urethra, Catheterism, and False Passage" (founded on post-mortem examinations), in which he shows how very frequent at that time was the occurrence of false passage in patients who died under treatment for stricture. In Case 5, for instance, "there were four ways of entering the bladder besides the right one." It is to be hoped that no such case could ever come to light at the present day, but the improvement in treatment which has taken place since then is mainly due to Mr. Cock's persistent advocacy of soft instruments and gentle dilatation. This appears to have

been his first essay on a class of cases, in the treatment of which he afterwards gained such wide-spread renown.

In 1852 he read before the Royal Medical and Chirurgical Society a paper founded on forty cases of puncture of the bladder through the rectum, all of which had come under his own observation. This was introduced by an eloquent declamation against the vicious practice of forced catheterism and other violent measures for the relief of impermeable stricture, which were then rife, and which, from time to time, are certain to be revived. Indeed, his life-long practice was a continual protest against the rough and clumsy wounds and contusions that are accounted tolerable in some schools of surgery. He revived the operation of puncture *per rectum*, with improved instruments designed by himself, as one easy of performance, and thus within the reach of any practitioner who is brought face to face with a case of retention where he and others may have failed to pass a catheter. He speaks with indignation of the practice which glutted the shelves of the museums with preparations, in which the urethra was found "bruised and lacerated, pierced at all points by the introduction of instruments which have effected a passage in every direction but the right; which have entered everywhere except at the narrow strictured opening through which they were intended to pass." He terms "forced catheterism" an operation Alexandrine in character, and thus describes it:—The surgeon takes "a small but strong catheter, one that will not easily bend, and, firm of purpose, with unflinching hand and fearless—I had almost said remorseless—heart, he carries it through all intervening structures and obstacles into the bladder. It is, as it were, puncturing the bladder with a blunt instrument—*tunnelling* through the perinæum under cover of the urethra. It is neither scientific in its conception, surgical in its performance, nor anatomical in its details. It can only be countenanced and justified on the principle that the end sanctifies the means. Attempts have been made to modify and soften down its essential roughness by denominating it the operation of forcible dilatation or breaking down of a stricture, and the surgeon, perhaps, tries to delude himself into the idea that his instrument either enters the stricture and

forcibly expands it, or else that it breaks it down and destroys it.

"To the best of my belief neither of these desirable events occurs. The catheter neither expands nor crushes the stricture, but it altogether eludes it, passing outside the obstacle, and then either re-enters the urethra below, or else continues its course through the prostate to the bladder. I have, at different times, had an opportunity of examining (post mortem) the urethras of many persons who had evidently undergone this species of catheterism, and I have never failed to discover the line of road, or, in railway parlance, the loop where the instrument had left the canal to re-enter it at some point below the stricture." The paper includes a reference to the experience of Mr. Hilton, his colleague, who had then punctured the bladder *per rectum* eight times. "The old stricture case (W. W.)," writes Hilton, "makes the eighth. It was there I had the advantage of your experience and opinion, and your aquiescence to justify the proceeding of puncturing the bladder *per rectum* for the cure of a very obstinate and irritable stricture. The result was as favorable as possible; the patient became quite free from stricture. I must, in conformity with my feelings, congratulate you as a surgeon, for having promoted by your hospital practice the revival of a most useful but much neglected operation on the urinary bladder." This paper, no doubt, did much to throw Mr. Cock's practice in the direction of a speciality, for practitioners learnt by it that he was a surgeon to be trusted always to use the gentlest means but sure, in the treatment of an affection which the malpraxis of the day had too often relegated to the domain of quackery.

In the thirty-fourth volume of the same Society's 'Transactions' Cock publishes an interesting case of arterio-venous aneurism following a puncture of the popliteal artery and vein, for which the femoral artery had been ligatured eleven years before. The sac suppurated and was treated by amputation through the thigh. The after-course reminds one of a past era in surgery before the introduction of antiseptics, for we read "in a month's time the stump had nearly healed, and I considered him as convalescent. He then became the subject of low fever, accompanied by bronchial irritation and

general constitutional irritability. His appetite failed, and he began to exhibit all the symptoms which had preceded several cases of fatal phlebitis, a disease which was at that time epidemic in our hospital." Cock seeing the danger had his patient removed to the country, where he rapidly recovered; and he afterwards became gate-keeper at the Custom House. Students of the present day can scarcely appreciate the change that has taken place as the result of Listerism. They are ignorant of hospital gangrene, of phagedænic ulceration, of secondary hæmorrhage, and of those terrible rigors which ushered in pyæmia,—then the suppurating joints, the pulmonary embolism, and lung-infecting abscess. All this belongs to a past age, and I was probably witness of the last epidemic of hospital gangrene that will ever occur in Guy's Hospital, when Mr. Cock's dresser in Naaman Ward. It began in a case of amputation for epithelioma and spread rapidly, destroying the scrotum and skin on the pubes, ending fatally in a few days. After this, every wound in the ward took on an unhealthy action and began to spread or slough. Cock tried burning the edges of the wounds with fuming nitric acid with partial success, but the ward had to be cleared and disinfected. To apply the acid Cock used pieces of cane or wood tapered like a pencil to a point and broken up at the end into a kind of brush. He did not find one ready for use on a certain occasion, and remarked rather testily that a bundle ought to be kept in the ward always at hand. A convalescent patient took the hint. He made a bundle, and, going round to Mr. Cock's house when he knew that surgeon must be engaged in the hospital, told Mr. Cock's servant that his master had told him to bring this parcel and that he was to give him a sovereign for it. This the servant was weak enough to do, and when Mr. Cock returned he found his servant had paid a pound for a trumpery little handful of tapering sticks.

Mr. Cock was a bold and skilful operator, always ready to adopt a new procedure, and possessed of a great deal of natural resource. He was the first surgeon in this country successfully to perform the operation of pharyngotomy for the removal of an impacted tooth-plate from the gullet. This he did in 1856, and the case is published in the 'Guy's

Hospital Reports' for 1858. A second case, also successful, he operated upon some years later for a similar accident, and this is related in the 'Guy's Hospital Reports' for 1868. The only ill-result which followed was a certain alteration in the voice, which the author attributes to division of some of the fibres of the recurrent laryngeal nerve. On reading the cases I note that the superior thyroid artery was divided, and I think it more probable that the external laryngeal branch of the superior laryngeal, which lies beneath the artery, was the nerve damaged, so that one crico-thyroid muscle became paralysed. A lowering in the pitch of the voice was the change particularly noted, so that a "fine tenor voice" was converted into a "respectable bass."

Another paper, published in the 'Guy's Hospital Reports' for 1842, "On two Cases of Injury to the Head" shows Mr. Cock as a practical and scientific surgeon in the most favourable light. One of these was a case of middle meningeal hæmorrhage, for which, when symptoms of compression occurred, Mr. Cock trephined, though no fracture could be discovered either before or after trephining. The operation was followed by immediate relief of symptoms, and the patient made a complete recovery. This is one of the few cases, and among the earliest, where trephining for blood has been successful. The patient lived fifteen years after, and died under Sir W. Gull's care of apoplexy in 1855, the sequel being reported in the volume for 1857. The second case recorded is one of suppurating cerebritis following fracture of the frontal bone, for which Mr. Cock removed with Hey's saw a portion of the bone injured, and then divided the dura mater and probed the brain to allow the escape of pus. This case also was followed by a satisfactory result.

Among Mr. Cock's other contributions to the 'Reports' are no less than four papers on "Obscure and Difficult Forms of Hernia," which occur in successive volumes; and in conjunction with Dr. Hughes a paper was published in 1844 on 'Paracentesis Thoracis,' which at that time was an operation rarely performed. Both Dr. Hughes and Mr. Cock agreed in regarding the operation as one of no serious moment, and one usually followed by relief of symptoms and often by cure. Mr. Cock alludes to a very fine trocar and canula invented by

Dr. Babington, which he used as an exploring needle just as we use a hypodermic needle and syringe at the present time to make certain a doubtful diagnosis. It will be seen again that Mr. Cock and his corresponding physician were far in advance of the practice of the day in regard to the surgery of the thorax.

In subsequent volumes are papers on fractures and dislocations, in which it appeared expedient to divide the tendo Achillis for the purpose of replacing the separated bones and retaining them in position; rare cases of fracture and dislocation; on a case of myeloid tumour of the scapula weighing a pound, which Mr. Cock successfully removed with the spine of that bone and part of the clavicle; on select cases of aneurysm; and on mutilation of the forearm. His last paper, published as late as 1884, on surgical reminiscences, is an interesting one, and contains an account of the post-mortem examination he made in 1843 on Mr. Drummond, Secretary to Sir Robert Peel, who was shot in the back by McNaughton, in mistake for the principal. The course which the bullet took was most remarkable, for it crossed the pleural cavity without wounding the lung, and traversed the abdominal cavity without injuring a viscus, being extracted in front from beneath the skin between the seventh and eighth costal cartilages.

But the paper that has attracted most attention, and the one upon which his posthumous fame will chiefly rest, appeared in the 'Guy's Reports' for 1866. In it he describes a method of opening the urethra for impermeable stricture, "the last resource available in certain cases." The essay itself is a masterpiece of excellent writing, fluent, clear, and incisive; and in its completeness reminds one of those three pages in the 'Edinburgh Medical and Surgical Journal' of 1814, by which Colles spanned the mighty chasm which intervenes between intellect and immortality. The operation of opening the urethra behind the stricture was not a new one. It had been practised in a way by French surgeons of the last century, under the general term of *boutonnière*, and was mentioned both by Astley Cooper and Brodie in their writings as a proceeding which might in certain cases be adopted. Moreover, in 1852, Mr. Simon, now Sir John Simon, published a lecture in which he recommended a similar operation to be

very generally adopted for impermeable stricture, whether the bladder was distended or not. Seeing, then, that actual precedence is not with Cock, and that the origin of the operation is buried in the past,[1] on what foundation can the credit of it be associated with his name? The answer to this question will be given under three heads. 1. He developed the operation, and described in detail a method of performing it which differs essentially from that recommended by Sir John Simon, upon which it is a great improvement. 2. He pointed out precisely the class of cases, for which it was not only advisable but absolutely essential, and thus defined the position it should occupy in surgery. 3. He alone recommended, in certain appropriate cases, the establishment of a permanent fistula in the perinæum, or the formation of a "Cock's urethra."

1. The details of the operation he thus describes:—"The only instruments required are a broad double-edged knife, with a very sharp point; a large silver probe-pointed director with a handle; and a canula, or a female catheter, modified so that it can be retained in the bladder. The patient is to be placed in the usual position for lithotomy, and it is of the utmost importance that the body and pelvis should be straight, so that the median line may be accurately preserved. The left forefinger of the operator is then introduced into the rectum, the bearings of the prostate are carefully examined and ascertained, and the tip of the finger is lodged at the apex of the gland. The knife is then plunged steadily but boldly into the median line of the perinæum, and carried on in a direction towards the tip of the left forefinger which lies in the rectum. At the same time, by an upward and downward movement, the vertical incision may be carried in the median line to any extent that is considered desirable. The lower extremity of the wound should come to within about half an inch of the anus. The knife should never be withdrawn in its progress towards the apex of the prostate, but its onward course must be steadily maintained until its point can be felt in close proximity to the tip of the left forefinger.

[1] "Speaking of it as a mere anatomical operation, one could, perhaps, not easily name the time when it was unknown." Sir J. Simon, 'St. Thomas's Hospital Reports,' 1879, p. 140.

When the operator has fully assured himself as to the relative positions of his finger, the apex of the prostate, and the point of the knife, the latter is to be advanced with a motion somewhat obliquely either to the right or to the left, and it can hardly fail to pierce the urethra. If, in this step of the operation, the anterior extremity of the prostate should be somewhat incised, it is a matter of no consequence. . . . The knife is now withdrawn, but the left forefinger is still retained in the rectum. The probe-pointed director is carried through the wound, and, guided by the left forefinger, enters the urethra and is passed into the bladder. The finger is now withdrawn from the rectum, the left hand grasps the director, and along the groove of this instrument the canula is slid until it enters the bladder."

The following is Sir J. Simon's description:[1]—"Often in thin subjects, and where the urethra has not given way, the distension of this canal, as the patient strains to make water, will render it so evident, that your operation resolves itself into a mere puncture with a lancet. In the less easy cases, where your subject is fat, or the perinæum deep and infiltrated, there is really nothing to deserve the name of difficulty. You make a sufficient cut in the raphe, terminating a little in front of the anus, and sinking as deep as may be requisite into the cellular tissue. You may then, in the following way, arrive at the point of the urethra which you wish to penetrate. Pass your right forefinger into the anus; ascertain through the wall of the bowel the position of the prostate; bring your finger forward till it discovers the anterior extremity or apex of the gland; let it just pass this spot, and rest (nail upwards), pressing with its point immediately in front of the gland. Of course, if the parts were transparent, you would now see your finger indenting the membranous portion of the urethra at that hindermost point of its course where you propose to puncture it. Now pass your left forefinger (nail upwards) into the wound; advance it till (with the guidance of the finger in the rectum) it falls against the apex of the prostate; there you so arrange it that the middle phalanx presses back the rectum; the last phalanx lies along the prostate with the tip of its nail indicating the spot at

[1] Op. cit., p. 160.

which the urethra emerges. Finally, withdrawing your right forefinger from the anus, and resuming the bistoury, you run this along the left forefinger till you penetrate the canal on which it rests, and immediately follow it by the short elastic catheter which you intend leaving in the bladder."

It will be seen, with the exception that the goal reached is the same in both cases, there is nothing in common in these two operations. Simon makes no mention of the importance of the left forefinger in the rectum, and of its retention there, as a guide, till the bladder has been reached. His operation, in simple cases, is a "mere puncture with a lancet;" in difficult, he describes a somewhat clumsy roundabout way of finding the urethra, during which the knife is laid aside, and both forefingers are engaged at the same time, the right being in the rectum, the left in a preliminary perinæal wound. Cock's operation is as different from this as a railway journey to the old coach road—as distinct, as a telegraphic route is to the lumbering parcels' post. Let those who would claim the operation so practise it; but let them not detract from the value of Cock's canal till they have towed their boat in the wake of his canoe.

2. In the lecture referred to, Sir J. Simon recommended the perinæal operation indiscriminately; for all cases of impermeable stricture where it is essential to reach the bladder without delay; where, from the state of the patient, there is no time for gradual dilatation of the stricture; and for cases complicated with extravasation. This lecture is illustrated by six cases, five of which were for extravasation of urine, and for the sixth he curiously enough inserts the following apology:—"It is true that his urgent symptoms would have been relieved if I had tapped the bladder above the pubes or by the rectum; and his case, in my judgment, is that one of the series in which it most nearly became a matter of indifference which of these operations should be performed." Now this is the very case of the series, a man with a scrotal fistula, many strictures, and in a typhoid condition, that Cock would have said the operation was especially adapted for. To relieve cases of extravasation the operation, if advisable (and I usually myself employ it), is not essential; whereas a free deep incision into the perinæum

alone at once relieves the patient and places him out of immediate danger. I wish I could find space to quote Cock's eloquent description of the helpless, hopeless condition of the poor wretches for whom he especially recommended this operation; but, shortly, they were those cases in which the bladder was so contracted as not to admit of puncture, where the strictures were impermeable, and the scrotum and perinæum riddled with fistulæ. In these cases it remains the "last resource available." It can never happen that this operation should entirely replace simple puncture for distended bladder with impermeable stricture as recommended by Simon; firstly, because it is more difficult; secondly, because it is more dangerous. I have myself performed it in a very large number of cases, and have completed it, on one occasion, in eighteen seconds. Once I performed it, at Mr. Cock's suggestion and in his presence, without chloroform; yet only the other day, in a case where the urethra was pushed altogether on one side by a perinæal abscess, my knife in the first incision completely missed it, and it was not till I made further search that I discovered the passage, bare but unopened, on the right of the abscess cavity. An inexperienced operator, without assistance, would, under such circumstances, I feel convinced, fall into confusion and give up the operation. Useful as the operation is in a special class of cases, for retention it can never become universal.

3. Mr. Cock was the first surgeon to suggest that after the completion of the operation, where the urethra is irretrievably damaged, a permanent canal should be retained in the perinæum, so that micturition might become assimilated to that of the other sex. He alludes in his paper to some half a dozen patients, happy and comfortable, on whom he had operated, using permanently this artificial canal through the perinæum. I could point to quite as many among my own patients, and one of these I operated on for Mr. Cock when he was ill, nearly twenty years ago, and the gentleman still remains in a perfect state of health. The credit of this extension of the operation is, I believe, entirely due to Mr. Cock, and it is a point, I think, which has never been disputed.

In thus endeavouring to show the grounds which, in my

opinion, clearly justify the association of Cock's name with this operation, I should feel deeply grieved if I thought I might thereby cause pain to so accomplished and excellent a surgeon as Sir John Simon. There are few in this generation who have held so high a scientific position, and for whom I have so great a reverence, but in my attempt to do justice to my former colleague and teacher, I feel I can have said nothing that will detract from the reputation of another, who among the great is among the greatest.

In appearance, Cock was a man rather below the average in stature, and, had it not been for his handsome face, his figure would have attracted little attention. He had, moreover, in early life a slight stoop which made him appear prematurely old, but the familiar expression "old Teddy Cock" was probably more a term of endearment than one referable to his age. His grand head and face and pleasant expression, however, atoned at once for a somewhat diminutive frame. A delicate pink complexion, a good but not excessive forehead sloping gently to a pair of arched brows, a full, tender blue eye protected by gold-rimmed spectacles, a firm, well-chiselled aquiline nose, and a most attractive smile, together created an impression not easily forgotten. His white hair remained thick to quite old age, and curling up at the end gave him a somewhat gallant air, whilst his short beard being confined to lips and chin left the dimples of his rounded cheeks exposed in laughter. A few words with him and you found you were in the presence of a man of high culture possessed of an infinite sense of humour.

In dress he was peculiar and quite indifferent to all conventional ideas of professional attire. In summer he usually wore a rather large shepherd's plaid for trousers, a buff waistcoat with onyx buttons, and a silk-lined jacket, with a blue-spot necktie and low crowned soft felt wide-awake hat. In winter the white hat was changed for a black one of the same shape, and the silk fly-jacket was replaced by a pilot coat. A massive gold chain, a silver snuff-box, and an ivory-handled cane complete the picture.

His peculiar attire was probably that which experience had taught him was most comfortable to wear when on horseback, for Cock commenced practice at a date when it was usual for

consultants as well as general practitioners to work their practices largely by the aid of saddle-horses—before the days, indeed, when an advanced and humane civilization had discovered that an irregular intermixture of greasy asphalte, unyielding blocks of stone, and uneven puddle-hollowed wood pavement was the contrivance best adapted for the destruction of horses' legs, and for the prevention of their retaining the erect position. Although this change from the old Macadam road caused Cock in later years to take refuge in the cumbersome brougham, he still continued to ride between his country house and the station morning and evening, and to enjoy long rides in the country lanes, or to join in the invigorating sport of following the hounds. Cock was a great lover of horses and made them his pets and companions. He always preferred mares to geldings, as he said they were more natural in their affections and possessed more human nature. At one time he owned a very vicious mare he named Jezebel, which was all gentleness to him but spiteful in the extreme to strangers. He would take his friends to see her in the paddock, and introduce her as a virgin of extreme prudery, then suggest they should address her by a title reflecting on her modesty, whereupon the mare would put back her ears and fly at them open-mouthed. Cock's sense of humour was infectious, and seemed to spread even to the animals about him. Another favourite mare named Polly carried him for years, living in luxury and dying with honour. His single-horse brougham was always drawn by a fast-trotting horse, and at one time was perhaps the best known in town, being decorated with a cock rampant, and the motto "Whilst I live I'll crow."

At the beginning of this century snuff-taking was the prevailing habit, which smoking gradually replaced, and Edward Cock was to the end of his days an inveterate snuff-taker. There are many tales concerning his snuff-boxes which show that his kindness was such as to make thieves honest men, and would-be murderers grateful patients.

On one occasion, when engaged among the out-patients, he had remarked to his class that losses through pick-pockets were commonly due to the loser's carelessness and stupidity. Before he had finished he felt for his snuff-box and found

that it had vanished. He went home after expressing himself rather strongly on the event, and shortly after a person called and was introduced as a patient. When Cock handed him a prescription this shabby-genteel individual, to Cock's great surprise, produced his snuff-box, which he said he had called to return. "But how on earth did you do it?" exclaimed Cock. "It is very easy," said the thief, "when you know how," then proceeded to admire the pictures on the walls, the beauties of which Cock was tempted to explain. After this he bade farewell, but turning back at the door, to Cock's utter amazement, handed him again his snuff-box, which he had possessed himself of for a second time.

In the year 1886, one of his colleagues on the Kingston Bench of Magistrates, Mr. Philip Jones, received as a present from Mr. Cock a snuff-box made out of oak, which had lain 656 years in the foundation of old London Bridge, and was removed with the bridge in 1832. Accompanying it was sent the following interesting history in Mr. Cock's handwriting:—"Many years ago a Mr. Overstone kept a shop of curiosities near Soho Square. He was considered to be eccentric, and sometimes excitable beyond the limits of ordinary eccentricity, but devoted to his business. He made an expedition to Scotland of some extent, in search of curiosities, forgetting to pay his rent, which had been due a considerable time; and when he returned found that his landlord had put an execution into his house and advertised his furniture and goods for sale. Exasperated beyond bearing, he loaded a pair of old pistols with ball and probably a very small quantity of powder, and went to the counting-house of his landlord in the City. A wrangle ensued, which ended in his firing one of the pistols into his landlord's face (the ball lodged in the upper jaw just above the molar teeth, whence it was subsequently extracted and no permanent damage ensued). He then put the other pistol into his mouth and fired it. Subsequently he came under my care. There was evidently no serious injury, and we were puzzled to find what had become of the bullet. The inside of the mouth was much swollen, and the passage of the ball was not visible, but by tracing a red mark on the face upwards to the head the ball was found just under the skin at the top of the head, and easily removed.

He recovered, and in the course of time was tried at the Central Criminal Court for attempt at murder. The jury, however, acquitted him on the score of insanity. On the evening of the trial he sent me the snuff-box, with a short note, saying that, although a sapient jury of his countrymen had pronounced him insane, he was not so mad as to forget the great kindness I had shown him, &c., &c., although it perhaps would have been better if I had allowed him to die."

The dominant traits in Cock's character were a tender sympathy with suffering, and a generous warm-hearted benevolence. Unlike many who have risen to eminence in our profession, he did not gradually shut out the poor and retain only the wealthy as his patients. He practised to the end of his days in a narrow street in the immediate vicinity of the hospital, selected not for the convenience of the rich, but that hospital patients might the better gain access to him for advice, and reap the benefit of his wide experience. Although practising in such an unostentatious way, that his reputation alone served to attract the wealthy from a distance, his practice for many years was upwards of £4000 per annum. Somewhat petulant and hasty, his face would flush with indignation at anything like uncharitableness or unkindness towards a poor man or an outcast from society. Thieves and vagabonds and those who form the froth of the most polluted streams of human life, all knew that tender-hearted Cock would not judge them harshly, and would do his utmost to relieve their sufferings. The extent to which he carried these principles would raise a curl on the lip of many a modern Pharisee. He always had a good word for everybody, and tried to see only the best side of a man's character. I have often heard him say, for instance, that it was a mistake to suppose that an immoral man was wholly bad. He might be a patriot, and a most honourable man of business. In this, to some extent, I am inclined to agree with him. Immoral men make excellent politicians, soldiers, sailors, and judges; but very bad doctors, and only indifferent clergymen. Himself, a Conservative and a Churchman, ever ready to assist in philanthropic schemes, he looked askance at amateur preachers and at medical missions, seeing that they were too often used by hypocrites for the sake of adver-

tising personal holiness, or for the baser motive of gaining lucrative practice.

The short account given of Mr. Cock in the 'History of Guy's Hospital' by Dr. Wilks does scant justice to him as a surgeon, but contains a truthful record of his generosity and benevolence. It is, indeed, the picture of a surgeon seen through myopic medical spectacles, whereby everything of surgical importance has to be reduced in size to be perceptible to a physician's ken. But an anecdote there related of a particular instance of Cock's mode of relieving the outcast is so well told that I must fain subscribe the whole story. "A patient who had all the front of his face eaten away so as to leave a large hole in the middle of his countenance, was obliged in consequence to give up his post as gardener to the Greenwich Hospital. Mr. Cock took compassion on him, fitted him up with a new nose and spectacles, and thus embellished, he subsequently became a well-known, though not an inviting, object to the inhabitants of St. Thomas's Street. When thus improved Mr. Cock informed him that he should be his gardener. There was then, as now, a plot of ground, consisting of a few square feet, in the rear of St. Thomas's Street, where once stood, we believe, Mr. Churchill's bookseller's shop. This was covered with grass and had a fountain in the middle, in which a live eel disported itself; a fig tree grew against Mr. Cock's house, and beneath it was a seat on which the surgeon could repose; a few flowers and shrubs completed the rustic picture. Mr. Cock gave the man weekly wages and fed him at his own house. In the early summer morning the old gardener might be heard merrily whetting his scythe, and during many weeks managed to excite a rustic feeling in the minds of Mr. Cock's neighbours by various innocent devices—by keeping the grass plot closely shaved, continually scraping the flower-beds, putting in the ground flowers full blown, and sowing seeds which never came up, he managed to get through a day's performance. Whether Mr. Cock or the gardener believed most in this Arcadian illusion we cannot say, but we do know that both master and man were made happier by it."

Cock was a gentleman to the back-bone, candid and outspoken, open-hearted and open-handed, easily flushed to

crimson indignation by discourteous acts or underhand dealing, a hater of all that savoured of cant or hypocrisy, and in himself a pattern of uprightness and good-fellowship. He would never allow a person to traverse a gentleman's word in his presence; and when at my final examination at the College of Surgeons I had to thank him for his protection. Mr. J. Lane and Mr. Hancock at the first table had examined me chiefly on aneurism, on which I was well posted—"We give you full marks; you could not have done better," said the genial Mr. Hancock as the bell rang. Somewhat excited by the compliment, I was received in the most distant way at the next table by that most orthodox surgeon, Mr. Le Gros Clarke. I at once fell foul of his pet theories by suggesting that the so-called ligamentum teres of the hip-joint served no use as a ligament. He next asked, "In what position must be the patient to acquire a dorsal dislocation of the hip?" Never having considered the question, I fell back upon a case, and related how a man, descending from the front of an omnibus in motion, was rolled between the vehicle and the wall of the Half Moon Yard, and was picked up with a dislocated hip. Mr. Clarke expressed a doubt as to the possibility of dislocation being thus caused, when Cock, who was marking, turned crimson, tossed his papers on the table and said, "Sir, how can you doubt Mr. Lucas's word? This was a case of my own, and I remember the history was precisely as he has stated." I had few questions after this outburst, but left, feeling certain that my reputation was in safe keeping.

A Dutch student, who afterwards changed his name, and who did not mind admitting that he was prone to make exaggerated statements, used to relate the following anecdote of his examination. He was asked the treatment for hæmatocele, and said in reply he would draw off the fluid by means of a trocar and cannula, after which the fluid would not recollect. "And how many have you seen cured in this way?" asked the examiner. "I have treated six cases in this way myself," said the candidate, nothing daunted, "and they all recovered." "There," said the examiner triumphantly, "What do you think of that, Mr. Cock?" "I think it an exceptional experience," said Mr. Cock, "but the g— g— gentleman says he has seen them."

In 1858 Mr. Cock experienced the misfortune (to which operating surgeons are very liable) of receiving a poisonous inoculation on the hand, which was not only followed by severe local symptoms, but by constitutional disturbance of so distressing a character that his health became completely undermined, and his nervous system so shattered that he contemplated throwing up his appointments and retiring from the profession. It was then he learned the value of a true friend, whom he found in his old student-companion, W. Sudlow Roots, of Kingston. This excellent surgeon and genuine friend invited him down to his own house, there to remain until his health might be recruited, and I have heard from one who witnessed the scene of the pathetic meeting which then took place:—"I am come, my dear friend," said Cock, "at your invitation, to die in your house." Not so—the good Samaritan, not content with taking him into his house and supplying him with the best of food and wine, mounted him on his own horses, and sent him out, with his only son as a companion, to enjoy the air and partake of the care-forgetting delights of the chase. For two years Cock remained a guest in Mr. Roots's house, travelling daily to town as soon as his health permitted, and returning to sleep in the country at night. After this he went to live in one of Mr. Roots's houses at Townsend, Kingston, and there he remained till his death.

Being thus by illness introduced to country life and country sports, Cock from this time onward used his town house only for the purpose of seeing patients, whilst he gradually added to his interests in the vicinity of his suburban home. He became surgeon to the 12th Surrey Rifle Volunteers, and did much to encourage accurate shooting by liberal donations to the prize fund. There, mounted on his favourite charger, he might be seen at inspections and on field days ready to assist in the relief of accident or sudden emergency, and any member of the corps requiring operation was sure of his surgeon's kindest sympathy and best attention. It is needless to say that he became the most popular officer in the regiment.

Again, when a local hospital was started, as a matter of course Mr. Cock was elected its consulting surgeon; and in 1875, at the request of the leading men of the town, he

allowed his name to be submitted to the Lord Chancellor as a fit person to be put on the commission of the peace, and he was duly appointed a Justice of the Peace. In that capacity he was most attentive to his duties, never failing to attend when on rota, but his influence was always exerted towards the lenient side, so that prisoners were regarded fortunate to be tried when Cock was sitting on the bench. I was told by a resident in Kingston, who attended the funeral, that it was Cock's frequent habit, after sentencing a man to be fined for some trivial misdemeanour, to slip round to the back of the court and drop the fine into the prisoner's hand, at the same time telling him not to commit the offence again.

The same kindness of heart characterised him as an examiner, and if any error of judgment ever occurred it is quite certain the candidate never suffered at his hands. A friend remarked one morning that he was looking haggard, whilst an examination was progressing. "I could not sleep last night," said Cock, "for thinking how I may have permanently damaged that poor young man's future whom I rejected."

The hounds he used to follow on his idle days were Garth's, the Surrey Union, and the Surrey Stag Hounds. The latter he soon gave up riding with, as he said such ungentlemanly fellows were to be found among the field. The particular event which caused him to take this view was a day when the hounds left off somewhere near Sutton, and a very plausible and well-dressed sportsman volunteered to show Mr. Cock the way home. He led him to the "Cock," at Sutton, where they had a sumptuous repast together. After dinner the courteous stranger said he would run out and see the horses were properly looked after. Finding he was a long time before returning, Cock made inquiries, and discovered that his unknown friend had decamped leaving him to pay for all that had been ordered.

His zealous attachment and devotion to the hospital where he was educated were almost unparalleled. During the years he was employed as surgeon and lecturer he was rigidly exact in his attendance, and never allowed private engagements or other interests to interfere with his public duties. After his retirement to the consulting staff, without ever volun-

teering his opinion, or attempting to force his views upon his juniors, he conceived it to be his duty to be within reach if required, and subsequently for more than twenty years he might be seen on Tuesdays and Fridays in the operating theatre, watching the operations and ready to relate his experience if solicited. Dr. Wilks's definition of a consulting physician to a hospital as "one never consulted" did not apply to Cock as a consulting surgeon. His friendship was so sincere that his advice carried no reservation, and the encouragement he would give to a young surgeon in trouble or doubt was heartily appreciated by his juniors.

About 1878 he began to suffer from rheumatism in his hands, which compelled him for a time to seek rest in retirement. He was advised to go to Buxton, and during his absence he asked me to take charge of the few patients he still retained. It would require the pen of a Dickens to describe the curious medley of old patients to whom he gave gratuitous advice. There was an ancient Bermondsey herbalist, with a long white beard and pale, solemn face, who had once fractured his ankle and had been under Cock's care in the hospital; he was a regular receiver of bounty because the result of the treatment had not been satisfactory to the surgeon.[1] Another was a retired four-in-hand coach-driver that rheumatism had thrown out of employment; a third had been employed in the manufacture of microscopes until his eyesight had failed him; a fourth was a hunch-back, who could obtain no regular employment. I found great difficulty in satisfying these various callers, and in distinguishing the paying from the gratuitous class. For instance, one day a very well-dressed man called and asked me to tap him for hydrocele. This I did to my own satisfaction, and I think to his also. He hesitated some time before leaving (revolving in his mind, I thought, whether I was worthy of the same fee he was accustomed to pay Mr. Cock), then turning round at the door, he said, "Mr. Cock always gives me a sovereign every time he taps me," and I found the usual relation of surgeon and patient suddenly reversed.

Mr. Cock remained a bachelor until the age of sixty-two,

[1] This patient eventually died under my care, and his ankle is preserved in the museum.

and then married Marianna, a daughter of the late Dr. Nunn, of Colchester. The story of this latter-day romance was related to me as follows; but I will not vouch for its literal accuracy. It is said that a friend meeting him one day exclaimed, "Why, Cock, how is it you are still a bachelor?" "Because nobody ever proposed to me," replied Cock with his accustomed humour, assuming an air of pitiable neglect. "Then I shall take the initiative," said the friend, "for your old sweetheart, Miss Nunn, has been waiting for you for the last twenty years, and I think it is time you married her." This friend is said to have invited the pair to his house, and to have in this way brought about the union. Mrs. Cock was a good-natured, intelligent person, about the middle period of life at the time of her marriage—one not soured by disappointments, not pinched into fastidiousness by maiden fancies, or hyperæsthetic from ill-health, but easy and kind-hearted, making Mr. Cock an excellent and devoted wife.

After his marriage the gentle attempts of his wife to correct some of his bachelor ways were often a great source of amusement to his friends. One evening it happened that he had invited two or three old friends to join him and his wife at dinner. As usual, before dinner was over, his favourite snuff-box had found its way from his waistcoat pocket to a place on the table beside his wine-glass. His wife seeing this, made an excuse to rise from the table when her husband was occupied, and stealing behind him took possession of the box. After a while Cock began mechanically to feel in his pocket and elsewhere for the missing companion, then gradually becoming conscious from the countenances of his friends and the smile on his wife's face that a trick had been played him, slowly put his hand into his tail-pocket, withdrew a large quarter of a pound paper packet of snuff, and, helping himself to two huge pinches, remarked to his wife, "Th-there, my dear, I think I have d-done you this time."

Though marrying so late, Mr. Cock had a happy married life of nearly twenty years, and was left a widower in 1886. During the last six years of his life, his house was managed with all kindness, gentleness, and consideration for his age, by the daughter of his old student-friend, Mr. W. Sudlow Roots, of Kingston. Like his distinguished uncle, Cock died

childless. So did Thomas Carlyle and the brilliant Earl of Beaconsfield, whilst the sons of the latter's great rival appear to illustrate Mivart's law of the necessary retrocession to an ordinary type. I have often thought what a misfortune it is that genius cannot be grafted, like tender fruit on the quince or paradise stock, or be budded like the rose on the stem of the wild briar. Until some such method is discovered, the fool of the family will be always first in the field, digging at the roots of the tree of reproduction to insure the continuance of his follies, whilst wisdom is turned aside, and knowledge is uprooted. Then genius, tender plant, exposed to wind and storm, shrivels and dies, leaving only the spirit of its past.

During his long life, Mr. Cock held, at various times, numerous public appointments, many honorary and some lucrative. Appointed Demonstator of Anatomy in 1825, he subsequently became Lecturer on the same subject, and continued to hold this chair until appointed full Surgeon. He was elected an Assistant Surgeon in 1838, was promoted to the senior staff in 1848, and retired to the consulting staff in 1871. When the Pupils' Physical Society budded off from the old Guy's Physical Society, Mr. Cock was elected its first Honorary President in 1836, and he continued to preside at the first and last meeting of the session for a period of forty years, where the anticipation of hearing his eloquent and warm-hearted speech of introduction at the beginning, or of congratulation at the close, always attracted an immense gathering. In 1849 he was appointed President of the Hunterian Society, to which honour no less than fifteen of the Guy's staff have at various times been elected.

From 1843 to 1849 he was one of the editors of the 'Guy's Hospital Reports,' an office for which he was particularly well suited, for his own papers are among the best worded of any that have appeared in this periodical. For many years he was consulting surgeon to the London and Brighton Railway Company. He was also consulting surgeon to the Asylum for the Deaf and Dumb, to the Philanthrophic Society, and to the Royal Medical Benevolent School at Epsom. He was elected a member of the Council of the Royal College of Surgeons in 1856, re-elected to that office in 1862, and retired in 1871, thus serving for a period of fifteen years. He

became a member of the Court of Examiners in 1867, but after serving five years did not seek for re-election. In 1867 he was elected Vice-President, again in 1868, and finally became President in July, 1869.

A remarkable event took place during his term of office as President, for the first meeting of Fellows and Members was held at the College on March 24th, 1870—a meeting which, owing to his tact and conciliatory manner, passed off most harmoniously. Mr. Trimmer, the courteous secretary of the College, who has kindly furnished me with the foregoing details, writes, "It is perhaps almost unnecessary that I should add, so far as his collegiate career is concerned, that he ever exhibited an intense interest in collegiate affairs, and was always most kind and considerate for others in the discharge of his several duties."

When he retired from his active duties as surgeon to Guy's Hospital, the past and present students subscribed to present him with a handsome silver épergne, as a token of their affection for him and appreciation of his services; and some years later his former colleagues defrayed the expenses of a portrait, which is hung in the hospital and is a good likeness, but taken too late in life, and by some exaggeration of expression suggesting—more than was ever true of him—the dethronement of intellect by age.

During the last two years of his long and useful life, which was extended to the patriarchal age of eighty-seven, his visits to the hospital he loved so well gradually ceased, and he lived in the quiet retirement of his Kingston home. But nothing gave him so much pleasure as to welcome a former colleague, and to hear from his lips what progress was being made in surgery, or what changes were being carried out in the Medical School. His warmth of affection never ceased, and his interest never dulled, but in the weakness of extreme old age he gradually faded away, and expired on August 1st, 1892. Few can hope to make so many friends, or to leave behind so little enmity, for all who ever knew him loved him; all who ever sought his advice were grateful to him; the students worshipped him; and the world, that was acquainted with his character, respected him.

# THE

# CAUSE AND PROGNOSIS OF ASCITES

DUE TO

# ALCOHOLIC CIRRHOSIS OF THE LIVER, TO PERI-HEPATITIS, AND TO CHRONIC PERITONITIS.

By W. HALE WHITE, M.D.

My object in writing this paper is to show that the commonly accepted explanation of the cause of ascites in cirrhosis of the liver will not always hold good, and that in spite of the recent publication of cases in which it has been considered that a cure has been effected, nevertheless the supervention of ascites in this disease is of the very greatest prognostic significance. So true is this that I have not, in consulting the records of Guy's Hospital for the past five years, nor in looking through the works of Murchison,[1] of Frerichs,[2] and Dr. F. Taylor's paper on "The Diuretic Action of Copaiba Resin,"[3] been able to find a single case of uncomplicated cirrhosis of the liver with ascites which has long survived paracentesis of the abdomen; not one has lived long enough for that opera-

[1] 'Lectures on Diseases of the Liver.'

[2] 'Clinical Treatise on Diseases of the Liver,' Syd. Soc. trans.

[3] 'Guy's Hospital Reports,' vol. xxxvi.

tion to become necessary a second time, and many have died before it became necessary at all. It is very important that fatal cases in which there was a post-mortem examination should alone be considered, for I shall be able to bring forward several cases which were, during life, considered to be examples of cirrhosis, and which were tapped oftener than once, but in each the diagnosis was after death shown to be either entirely wrong, or at any rate cirrhosis of the liver was not the only pathological condition present. Another point, therefore, which I shall try to prove is, that if a patient, who during life is supposed to be suffering from cirrhosis of the liver with ascites, has the abdomen tapped, but life is sufficiently prolonged to allow a second paracentesis to be necessary, the diagnosis of cirrhosis of the liver is almost certainly either completely wrong, or at any rate the patient has something else the matter with him in addition to his cirrhosis ; and further, it is highly probable that he has some form of chronic peritonitis, of which what is ordinarily called perihepatitis is merely a variety. These points will be considered separately ; and the most convenient order in which to take them will be firstly the prognosis of ascites, secondly its cause, and thirdly the cases in which cirrhosis was diagnosed, but paracentesis was performed oftener than once.

### 1. *The Prognostic Significance of the Ascites.*

In the first ten cases contained in the appendix to this paper ascites was present, but the patients died before paracentesis was necessary, and thus we learn that in them death took place before the amount of ascitic fluid had had time to become extreme. But it might be urged that this did not prove that the collection of ascitic fluid was a bad omen, for it may have collected very slowly. An examination of the cases in detail shows, however, that the reverse is true. In Case 1 it is not stated how long the ascitic fluid had been collecting, but it is very improbable that the time was long, for at the autopsy only two pints were found. In Case 2 the patient had noticed his abdomen swelling for four weeks before admission, and he died four weeks afterwards ; only

three and a half pints of fluid were found in the abdomen. From the date that fluid was first noticed till death was only eight weeks. In Case 3 the patient said he had only been ill for seven weeks before admission, and as the collection of fluid in the abdomen does not so frequently arrest the patient's attention as do sickness and symptoms of general ill-health, probably the fluid had not been collecting for so long. He died a fortnight after admission, therefore the time from the appearance of ascites till death was probably much less than nine weeks. In Case 4 it is stated that the abdomen had recently begun to swell, and the patient died sixteen days after admission. We may, therefore, fairly say that here also death quickly followed upon the supervention of ascites. In Case 5 it is not stated when the collection of ascitic fluid began. At the autopsy eight pints were found in the abdomen, and, judging by the other cases of ascites here recorded, this amount of fluid would not take long to collect. Case 6 had observed the abdomen to be swelling for four or five weeks previous to his admission to the hospital ; he died ten days afterwards, so that the time from his first noticing his ascites till his death was about six weeks. In Case 7 the abdomen had been swelling for a fortnight before the patient's admission ; he died five weeks afterwards, so that here the ascites was known to exist for seven weeks. Case 8 is that of a woman who died six weeks after admission, and of whom it is stated that the abdomen had been swelling for eighteen months before she came into the hospital. This makes the duration of the ascites at least eighteen or nineteen months. Every one knows how unreliable patients' statements often are ; therefore, as the duration in this case is so very much longer than in any others, I cannot help thinking there must be a mistake in the account given by the patient. In Case 9 the history is not sufficiently definite to enable us to fix the date of the supervention of ascites. In Case 10 ascites was first noticed four weeks before admission, and she died eight weeks afterwards, so that the ascites was noticed twelve weeks before death. If these cases are to be regarded as typical cases of cirrhosis—and I see no reason why they should not—we most certainly learn that the time from first noticing that the abdomen has begun

to enlarge till death is usually very short, probably on an average about two months.

Let us now see whether the cases 10—24 in the appendix, and in all of which paracentesis was performed, confirm this gloomy view. Case 11 had noticed the abdomen to be swelling for ten days before admission; he was at once tapped, and five pints were withdrawn, but he died two days afterwards, so that only twelve days elapsed between his noticing the swelling of the abdomen and his death, and the fluid probably collected at the rate of about half a pint a day. Of Case 12 it is stated that abdominal enlargement began fourteen days before admission. Two days after, twelve pints of fluid were drawn off; so that either a large amount of fluid collected before the patient noticed it, which is unlikely when we remember that patients have noticed their abdomen to enlarge when only two pints have been found after death, or, what is much more probable, the fluid collected at the rate of nearly a pint a day. He died nine days after admission, so that the duration from the first observation of abdominal swelling till death was only twenty-three days. Case 13 noticed the abdomen to be swelling for three weeks before admission; immediately he came into the hospital it was tapped, and twenty-three and a half pints of fluid were withdrawn. Here also it is highly probable that the fluid accumulated at the rate of nearly a pint a day. He died thirty days after the abdomen began to swell. Case 14 had first observed the abdominal distension seven or eight weeks before admission, and he died nineteen days afterwards. He was tapped a week after admission, and in his case the fluid did not collect so quickly; nineteen and a half pints were withdrawn, which gives a rate of collection of about a third of a pint a day. The duration from the first observation of the abdominal swelling till death was about ten weeks. The patient whose case is No. 15 was too ill to give an account of himself, and the only information we could get about the duration of the illness was hearsay. In Case 16 the duration from the time when the patient first noticed the abdomen to be swelling till death was twelve weeks. In her case the fluid did not collect with great rapidity, only six and a half pints being obtained

four days before death. Case 17 stated that his abdomen began to swell four months before admission, and he died a week after admission. In him also the fluid collected slowly. Cases 18 and 19 were two that were admitted for cirrhosis with ascites, and were tapped, and were discharged as relieved. The study of the other cases had so impressed me with the extreme fatality of cirrhosis with ascites that I wrote to the friends of each of these two patients to find out what had become of them. Each had died within a month of leaving the hospital, and each had been a heavy drinker; so, although there was no post-mortem examination in either, I think we may assume that in each case the patient really died from cirrhosis of the liver. I may here say that I have written to other patients who have been stated to have had cirrhosis with ascites, and who have been tapped and have been discharged as relieved, but I have had no answer from a patient in whom the diagnosis was probably correct except these two, and both died shortly after their discharge. Others, who will be alluded to later, had been readmitted into Guy's Hospital and died there, and it was found that the original diagnosis of cirrhosis was wrong.

To return to Cases 18 and 19: in the first the abdomen had been noticed to swell for three weeks before admission, and a week after she came in eleven and a half pints were withdrawn, which gives a probable rate of collection of just under half a pint a day. She died within eight weeks of the first observation of the ascites. In Case 19, eight weeks after she first noticed that the abdomen was swelling, twenty pints of fluid were withdrawn. She died about twelve or fourteen weeks after the abdominal distension first began. In Case 20 our data are insufficient; all we know is that the patient died twenty-eight days after twenty-four pints of fluid were withdrawn from the abdomen. In Case 21 the duration from the first observation of abdominal swelling till admission is stated to have been four months. He died three weeks afterwards, and four days after twenty-six pints were withdrawn from the abdomen. In Case 22 the time from the first observation of ascites till death was about six weeks. The fluid apparently collected at the rate of more than half a pint a day. In Case 23, from the first observance of

abdominal swelling till death was nine weeks, but the quantity of fluid removed is not stated. In Case 24 the abdominal swelling began three weeks before admission; paracentesis at once yielded sixteen pints of fluid, which gives a probable rate of secretion of considerably over half a pint a day. The patient died twenty-five days after the abdominal swelling was first noticed.

Of these fourteen cases, in twelve we have sufficient data to estimate the period between the time when the abdominal distension was first noticed and death. It was 12, 23, 30, 70, 84, 130, 56, 91, 140, 42, 63, and 25 days respectively in each case, giving an average of 63 days, or two months, which is the same average as we thought probable after considering those cases which were not tapped. These figures show the extreme danger of the supervention of ascites in alcoholic cirrhosis of the liver; and I have not been able to find a single case among the sources from which I have drawn those collected here, in any way showing that we can look for any other end than speedy death when once ascites has developed, for I shall later show that those cases which at the bedside appeared to disprove this statement turned out not to be cases of cirrhosis at all, and I shall mention those recorded by others which at first sight refute this view. It will be noted that occasionally, as in Cases 11, 12, 13, and 24, the patient was dead within a month of his first noticing that the abdomen was swelling, and that in only two cases was life prolonged beyond three months.

It may be asked whether paracentesis does any good in cases of alcoholic cirrhosis of the liver. This is very doubtful. When the embarrassment of respiration is great, the removal of the fluid seems to relieve the patient's distress for a time; but among the cases of uncomplicated alcoholic cirrhosis here recorded there is not one which shows that paracentesis does much good—nor, indeed, one in which the patient lived long enough for the fluid to re-collect in sufficient quantity to render a second paracentesis necessary; in fact, it may be laid down as a general rule that if the patient lives long enough for a second paracentesis to be required, either the diagnosis of alcoholic cirrhosis is wrong or it is only partially correct. In the latter case

it will be found that the patient has some other cause than the cirrhosis from which he suffers, which may help to explain his ascites. The number of days which the fourteen cases recorded at the end of this paper survived the first and only tapping was 2, 7, 9, 12, 11, 4, 8, about 30, about 40, 27, 4, a few days, 14, 5, in each case respectively. These figures certainly seem to show that paracentesis is of little benefit to the patient. If it had been we should have expected to find a few cases which had lived long enough for two or three tappings to be necessary, which had then succumbed, and in which nothing but cirrhosis of the liver had been found. It can hardly be that the operation is the cause of death, for the abdomen is often repeatedly tapped in the same patient in cases of heart disease, and in some cases of chronic peritonitis paracentesis is repeated ten, twenty, or thirty times. There is a woman who often comes to Guy's Hospital, whose abdomen has, up to December, 1892, been tapped 269 times. The true explanation of the fatality of the operation in cases of alcoholic cirrhosis of the liver is that supervention of ascites in that disease is of very bad augury, so that by the time the abdominal distension has reached a point at which paracentesis is necessary the patient is already near his end, which paracentesis can do nothing to avert.

That the mere presence of ascitic fluid causes death is unlikely, for many patients with alcoholic cirrhosis die before ascites becomes marked, its removal does not ward off the fatal end, and in patients in whom ascites is due to some other cause several hundred pints may be poured out before death closes the scene. It appears as though in cirrhosis of the liver the ascites and death had a common cause.

I have heard Dr. Fagge at the bedside express the opinion that the supervention of marked ascites in cases of cirrhosis meant that death was not far off, an opinion which is amply confirmed by the cases collected here; and he used to say that tapping the abdomen was the beginning of the end. The following quotation[1] from him also bears out this view :— "Moreover, when by means of diuretics and purgatives

[1] Fagge and Pye-Smith, 'Principles and Practice of Medicine,' vol. ii; and Fagge, 'Guy's Hosp. Rep.,' series 3, vol. xx.

one is able to clear the peritoneal cavity of its fluid, the successful action of such remedies seems often to avail the patient very little, for he presently becomes stupid and unconscious and dies, although his abdomen may be perfectly flat and empty. The question has even been raised whether the removal of the fluid by medicines may not have been sometimes concerned in the production of the fatal issue." So we see that it matters very little how the fluid is removed. The important point is that when there is enough to render its removal desirable the end is near. But, as has been just pointed out, seeing that ascitic fluid may be so often removed in other diseases with nothing but good to the patient, I should think it very unlikely that its removal in cases of ascites due to cirrhosis could accelerate death.

I have read what many authors say with regard to the prognosis in ascites due to alcoholic cirrhosis, and may give as examples the following opinions. Murchison[1] says of ascites in cirrhosis, "When once it appears, it persists and gradually increases." He also says, "Cerebral symptoms are a contra-indication to paracentesis, as they are usually aggravated by a withdrawal of the fluid." But I suspect that this is not quite the right way to put the statement, for I have already shown that probably by the time paracentesis is performed the patient is so ill that nothing can save him from death. Wickham Legg[2] states that the end often arrives soon after the tapping.

Frerichs[3] considers that paracentesis should only be performed when interference with respiration renders it necessary, because of the great exhaustion which must follow from the drain of such a large quantity of albuminous material from the blood when the fluid reaccumulates. But the cases to which I have already alluded, in which the ascites is due to other diseases than cirrhosis, and in which paracentesis is performed several times, are opposed to this view.

Several authors speak of tapping the abdomen frequently in the same case of alcoholic cirrhosis, and give instances of patients who after treatment in this way were discharged

[1] 'Diseases of the Liver.'

[2] Quain's 'Dictionary of Medicine,' vol. i, p. 836.

[3] 'Diseases of the Liver,' Syd. Soc. trans., vol. ii.

from the hospital much improved. Knessner,[1] for instance, used such a case for a lecture.

Dr. W. B. Cheadle[2] has quite recently published a very important paper upon the curability of ascites due to alcoholic and other forms of cirrhosis of the liver, in which he maintains the condition is curable. Certainly the evidence of the possibility of cure that he has got together is strong, and it may be that the cases I have collected are unusually severe. But in comparing the two sets of cases it must be remembered that some of Dr. Cheadle's cases—for example, the first—were syphilitic, and improved under iodide of potassium. Others, again, "recovered perfectly at the time, but were subsequently lost sight of." Perhaps, if it had been possible to follow them up, it would have been found that they had soon died. Some of the patients which I wrote to after they had been discharged after paracentesis were dead within a month. Then, again, much depends upon what we mean by the word cured: for instance, one of Dr. Cheadle's cases died of "a casual bronchitis" shortly after her discharge from the hospital; but from a pathological point of view she was not cured, for the cirrhosis was found post mortem, nor from her own point of view can she be said to have been cured. As Osler[3] says, ordinary cirrhosis of the liver is incurable. But it is always a pity merely to argue about words. My point rather is that if a patient has alcoholic cirrhosis of the liver with ascites it is in the highest degree improbable that he will live long; and the fact that one of Dr. Cheadle's cases succumbed to a chance bronchitis bears this out, for it is quite exceptional for adults to be carried off from a single attack of bronchitis, which, however, this woman was unable to withstand. The same argument also applies to his case who died from erysipelas. In the next place we have to remember the possibility that the diagnosis was incorrect. I have seen patients under the care of most skilful physicians—one I remember was seen by several—who have been thought certainly to have cirrhosis with ascites, but at the

[1] Volkmann's 'Sammlung klinische Vorträge innere Medicin,' No. 48.

[2] 'Brit. Med. Journ.,' Nov. 19th, 1892, p. 1102.

[3] 'Principles and Practice of Medicine.'

post-mortem examination there has not been the slightest evidence of cirrhosis. These cases are generally found to be examples of perihepatitis. As further showing the difficulty of diagnosis, it should be mentioned that at the very same meeting at which Dr. Cheadle read his paper Dr. Tyson[1] brought forward two instances of ascites for which no cause could be discovered. "These," he said, "were examples of a large number in which the ordinary explanations of the ascites were absent." Even the size of the liver is often wrongly made out during life; for example, in one of Dr. Cheadle's cases the organ was thought to be enlarged during life, but at the autopsy it was found to weigh only 36 ounces. Then there is another possible explanation of the temporary recovery of alcoholic cirrhosis with ascites, namely, that, although the patient has cirrhosis, the ascites is not due to this. We all know that cirrhosis is very frequently stationary, and presents no symptoms whatever; now the most common cause for cirrhosis is alcohol, and next syphilis. Perihepatitis, and the chronic peritonitis of which it is merely a part, are very often associated with alcohol or with syphilis. Hence it is very likely that sometimes a patient with a cirrhotic liver, which is itself giving no symptoms, may have perihepatitis and chronic peritonitis which are causing the ascites; and ascites due to this cause may often re-collect so slowly after tapping that some might, if they did not follow the case, regard it as cured. This explanation will very well apply to Cases 27 and 31 recorded at the end of this paper. I cannot help thinking it likely that Dr. Cheadle may have overlooked this explanation, for he says, "The only ascites due to chronic peritonitis of which I have knowledge is that set up by malignant disease of the peritoneum or tubercle." I cannot understand this; I always thought simple chronic peritonitis was a well-recognised disease.

Dr. Fagge,[2] as long ago as 1875, said, "Simple chronic peritonitis is very far from being a rare disease. In the hospital there is on an average one case of this kind to two of ascites from cirrhosis of the liver. It appears equally

[1] 'Brit. Med. Journ.,' Nov. 19th, 1892, p. 1106.

[2] 'Guy's Hosp. Rep.,' series 3, vol. xx, p. 204.

frequent in the two sexes. Of thirty-four cases that I have analysed, eighteen occurred in males, sixteen in females." He also describes it in his 'Principles and Practice of Medicine.' At Guy's Hospital, in 1888, the post-mortem records contain accounts of two cases, in 1889 of eight cases, in 1890 of two cases. I, however, very much wish that space had permitted Dr. Cheadle to publish full details of his cases, together with an account of the post-mortem examination when the patient died, for then no doubt many of my difficulties that I have felt while reading his paper would disappear. With regard to his four cases which are said to have absolutely recovered, time, with a post-mortem examination at death to see if the original diagnosis was correct, can alone show whether they ought to be regarded as instances of cure from cirrhosis.

Austin Flint[1] in 1863 published a very valuable paper on ascites, but the value of it is much diminished by the fact that among his causes of ascites he does not include simple chronic peritonitis. He says, "Undoubtedly this organ [the liver] is affected in the vast majority of fatal cases of hydroperitoneum;" but the quotation I have just made from Fagge, our experience in the deadhouse at Guy's Hospital, and some cases of perihepatitis I brought before the Clinical Society,[2] all show that chronic peritonitis is a frequent cause of fatal ascites. One point on which Austin Flint lays great stress, and which I have tried to emphasise, is that in cirrhosis of the liver there may be some other cause for the ascites than the cirrhosis. He gives twenty-two cases, none of them ordinary examples of cardiac or pulmonary disease, in which he thought this suggestion would apply. It is obvious that if in any one of these the patient had recovered from the ascites, it would not have been fair to call the case one of cirrhosis with ascites which was cured just because the presence of the fluid directed attention to the cirrhotic liver, which might very well have been causing no symptoms whatever. I may quote his words: "Cirrhosis of the liver being an incurable lesion, when the dropsy is dependent thereon recovery is hardly to be expected. It will be seen

[1] 'American Journ. of Med. Sciences,' vol. xlv, p. 306, April, 1863.

[2] 'Clin. Soc. Trans.,' vol. xxi, p. 219.

hereafter that although this lesion in all probability exists, an apparent recovery sometimes takes place; that is, the dropsical effusion may be removed, and a reaccumulation not occur for a greater or less period." We see how careful he is. In the cases that recover he does not positively state that cirrhosis was present at all; and even if it was, it may be that some auxiliary cause induced the ascites. But, as I said just now, Austin Flint's omission of chronic peritonitis greatly takes away from the value of his paper; and this is especially evident when we try to see whether we can find among his series a case of uncomplicated cirrhosis of the liver which required frequent tapping, for not only is there no account of a post-mortem examination in the majority of his cases, but even when there is we are not told whether chronic peritonitis existed. Taking all cases of ascites together, Austin Flint says that of his 46 cases 24 either ended fatally under his observation, or are known to have subsequently died. Of the remaining 22, in 8 there was no improvement when the patients were last heard of, "doubtless in most, if not in all of these cases the termination was fatal;" and after analysing the remaining cases he concludes by saying that "out of the 46 cases analysed, I can positively state that recovery has taken place in but a single instance," and this is a case of which he says, "circumstances which render it probable that cirrhosis does not exist." It is very difficult to harmonise this result with Dr. Cheadle's "7 to 8 recoveries out of 23."

Twenty years after, Dr. Flint[1] published some more cases of ascites, most of which were tapped, some oftener than once, but the subsequent history of many of these was only followed for a few weeks; in several cirrhosis was not even suspected during life. In only two cases was there a post-mortem examination: in one of these cirrhosis was found, but no ascites had existed during life; and the other was clearly an instance of chronic peritonitis and perihepatitis. This case was tapped eleven times. These cases, therefore, do not help us at all, nor do the four cases published by Dr. F. Roberts,[2] for in none of them was there

[1] 'Brit. Med. Journ.,' Sept. 22nd, 1883, p. 565.

[2] 'Practitioner,' vol. viii, 1872.

a post-mortem examination, and in two at least the diagnosis was obscure.

Bristowe[1] has recorded three cases of recovery from alcoholic cirrhosis of the liver. The jaundice and the alcoholic history in the first case render cirrhosis highly probable, but I do not think that in the absence of a post-mortem examination we can say that it was certainly present. I give examples at the end of this paper in which cirrhosis was diagnosed during life by skilful physicians, but at the autopsy it was found that none was present. The liver was not in this case ever felt to be enlarged. Then, as I have repeatedly urged, even if cirrhosis were present the ascites may have been due to some other cause. Dr. Bristowe speaks of her "many complications." She had rheumatic fever, bronchitis, and peripheral neuritis. And we must remember that persons who take to drink have often had syphilis. In the second case also there was no autopsy. The liver was never felt, but the ascites, jaundice, and alcoholism were certainly very suggestive of cirrhosis; but judging by the history the existence of syphilis was also highly probable; and what makes me especially think that possibly the ascites was not due to cirrhosis is that the patient had such extensive pleuritic effusion that paracentesis thoracis was necessary. This does seem to suggest that there was some common cause for the effusion into both serous cavities. In the third case evidence that the ascites was due to cirrhosis was very strong, but it is not quite conclusive, as there was no post-mortem examination. I, however, fully admit that the cases recorded by Dr. Bristowe are very strong evidence that ascites due to alcoholic cirrhosis is a condition from which recovery is possible; but it seems to me that, considering the many clinical fallacies, we cannot regard this as proved till we have had a post-mortem examination in some of these cases. One thing I feel quite sure of is that recovery is extremely rare.

Murchison also gives cases, but I cannot find recorded anywhere a case in which a post-mortem examination was performed upon a patient who had survived several tappings of the abdomen, and in whom the only cause for the ascites

[1] 'Brit. Med. Journ.,' vol. i, 1892, p. 847.

was cirrhosis of the liver. The published cases of recovery from alcoholic cirrhosis of the liver after frequent tapping almost prove too much, for they all get well; we have no intermediate cases which are tapped two or three times and then die. It is as though in patients with hæmoptysis one attack would prove fatal, but if the patient had more than one, recovery was certain. The last ten cases in the appendix to this paper (all of which were tapped oftener than once) bear upon the point under discussion, for in four of them (viz. Nos. 25, 26, 27, 34) the diagnosis of cirrhosis was made during life; but in three (viz. 25, 26, and 34) of these there was absolutely no cirrhosis, the ascites being due in one to colloid disease of the peritoneum, and in the other two to simple chronic peritonitis and perihepatitis: in the remaining one it was trifling, the ascites being clearly due to chronic peritonitis and perihepatitis.[1] So I suspect that some of the cases of cirrhosis that are described as discharged relieved or cured are really instances of simple chronic peritonitis. Perhaps in some others, in which the history of alcohol and the jaundice render cirrhosis highly probable, the patient had some cirrhosis of the liver, which it is well known may remain latent for years; but the ascites very likely was not due to this, but to chronic peritonitis, which is frequently associated with alcoholism. At any rate, until we have had recorded the post-mortem examination of a patient in whom the abdomen had been tapped more than once, and in whom no cause for ascites but cirrhosis of the liver can be found, I think I am justified in urging that the supervention of ascites in uncomplicated instances of alcoholic cirrhosis means that the end is near. Even when such a case has been recorded, we shall only have to alter this statement by inserting "almost certainly" before the word "near." So far as my cases go, they fully support the view of the past masters of clinical medicine, such as Watson, Niemeyer, Fagge, and others, as to the extremely grave prognosis of cirrhosis with ascites.

[1] While this paper was going through the press I saw two cases, both of which had been diagnosed as cirrhosis; one had been tapped many times, the other survived the only tapping for some months. Both proved to be instances of chronic peritonitis.

2. *The Cause of Ascites in Cirrhosis of the Liver.*

It will be observed that, in the cases already discussed eight or ten pages back, the fluid sometimes apparently collected at the rate of nearly a pint a day, and probably often at the rate of half a pint a day. This is difficult to understand if we believe that the slowly forming fibrous tissue is, by its compression of the branches of the portal vein within the liver, the sole cause of the ascites; for we know that a high degree of cirrhosis may exist without any ascites —indeed, it is quite common in the post-mortem room to find advanced cirrhosis of the liver in persons in whom it was unsuspected during life. In all cases of cirrhosis of the liver the excess of fibrous tissue must have existed some time before the ascites began; but as the cases here published appear to show that the fluid may suddenly begin to be poured into the peritoneal cavity at the rate of a pint a day, they do suggest that in some instances at least there must be an additional cause to determine such a sudden and rapid effusion of fluid. Perhaps it is due to thrombosis of some of the minute branches of the portal vein within the liver. I make this suggestion because it is stated that that very rare disease, pylephlebitis adhesiva, causes a very rapid accumulation of ascitic fluid, and because in Cases 1 and 15 and in other cases of cirrhosis an ante-mortem clot has been found in the portal vein. But against this view is the statement of Wilks and Moxon that "the check to the portal vein may in severe cases lead to entire stagnation of the blood in that vessel, with coagulation; and after that has occurred life may be prolonged at least *many weeks*, as the changes in the clot have proved." Still it must be remembered we often see in thrombosis of other veins that the effusion from the distal branches varies very much in quantity, and also in the rate at which it is poured out; these variations being due no doubt to the rapidity with which the clot forms, and to the proportion of the calibre of the vein which is blocked; for, as Dr. Goodhart[1] says, "sometimes the trunk is distended and completely occluded by a clot of large size, and more or less firmly adherent

[1] Dr. Goodhart, 'Syd. Soc. Atlas of Pathology,' fasc. iv.

to the wall of the vein; and sometimes only its hinder wall is covered with a flattened adherent decolourising clot." I should add that in some cases possibly the thrombosis does not occur in the branches of the portal vein in the liver, but in some of those anastomoses between the general venous and the portal circulation which are well known to be distended in cases of cirrhosis of the liver. Further, in support of the view that something more than the pressure by the cirrhotic tissue in the liver is required to explain the very rapid accumulation of ascitic fluid in some cases of hepatic cirrhosis, is the fact that even when the portal vein and other structures in the transverse fissure are pressed upon by lymphatic glands enlarged by deposits of malignant growth in them, the ascitic fluid does not collect more rapidly—indeed, often not so rapidly as in some cases of alcoholic cirrhosis. We must also bear in mind the possibility that the ascites which occurs in alcoholic cirrhosis of the liver is not caused mechanically, any more than is that met with in acute Bright's disease.

The swelling of the lower extremities, which so often supervenes in cases of cirrhosis of the liver, is usually set down to the pressure of the ascitic fluid on the inferior vena cava. But this certainly cannot always be the case. For instance, the case recorded by Dr. J. A. P. Price[1] was never tapped, and the account of the post-mortem examination says that "a small amount" of ascitic fluid was found after death; and, as still further showing that the ascites cannot have been the cause of the swelling of the feet, is the fact that the patient was quite positive that "on the night before admission his feet began to swell, then his legs and abdomen." But on admission no ascites could be detected, and so apparently the abdominal distension which he noticed the night before must have been due to tympanites and to his enlarged liver. Nor does it seem likely that the swelling of the feet can have been due to the pressure of this organ, which weighed 130 oz., upon the inferior vena cava, for it is noted that the blood in the superficial abdominal veins flowed in the normal direction. In a case of cirrhosis of the liver recorded by Dr. Carrington in the same volume of the 'Guy's

[1] 'Guy's Hosp. Rep.,' vol. xlii, p. 295.

Hospital Reports' it is stated that the patient's "legs and feet began to swell an hour or so after rising in the morning." No mention is made of any ascites either during life or after death, and the liver was not excessively large, for it only weighed 72 oz. However, the report repeatedly states that the patient was very anæmic, and no doubt anæmia is often the cause of the swelling of the lower extremities in cases of cirrhosis of the liver; but in Dr. Price's case this can hardly be so, because of the extreme suddenness with which it came on, and, although the report is very complete, no mention is made of anæmia.

Among the cases recorded at the end of this paper we find that in No. 3 there was œdema of the feet, but the ascites was never enough to call for paracentesis. Case 5 shows very well how suddenly the œdema may come on, for the patient said he first noticed his feet to be swollen two days before admission; the next day the penis was swollen, and on admission the legs, scrotum, and penis were very œdematous. These symptoms can hardly have been due to pressure upon the inferior vena cava, either by the liver or the ascitic fluid, not only because they came on so suddenly, but also because at death more than a month afterwards only eight pints of fluid were found in the abdomen, and the liver only weighed 65 ounces. In Case 8 the feet first swelled three weeks before admission, and this cannot have been due to the pressure of the liver, for this only weighed 47 ounces. Whether or not it was caused by the ascitic fluid or by anæmia one cannot say. The same remarks apply to the ninth case, in which the liver only weighed 43¾ ounces, and to the tenth, in which the weight was 36½ ounces. Case 12 is, however, interesting, for it is distinctly stated that the abdominal swelling and the œdema of the feet were noticed simultaneously, so that one of them can hardly have been due to the other, nor can the œdema of the legs have been caused by pressure of the liver, for that organ only weighed 60 ounces. In Cases 14 and 16 it is also stated that the swelling of the feet and the abdomen came on simultaneously. Case 22 is important, for it is stated that there was no swelling of the feet, and yet ten quarts of fluid were withdrawn.

So it is evident that extreme ascites may exist, and yet the fluid does not obstruct the circulation through the inferior vena cava. We see that while no doubt, in some cases of cirrhosis of the liver, the anæmia, or the ascitic fluid, or an enlarged liver will account for the œdema of the lower extremities, there are other cases which it is difficult to explain as due to any of these causes; and further, that sometimes the œdema of the lower extremities comes on very rapidly. This is suggestive when it is remembered that we have already shown how very quickly ascites may develope in these cases; and these facts seem to indicate that sometimes at least alcoholic cirrhosis of the liver is a much more general disease than is generally supposed. In confirmation of what I have here stated, it should be mentioned that Austin Flint,[1] thirty years ago, pointed out that the pressure of the fibrous tissue in the liver cannot in many cases be the cause of the ascites, for it may come on rapidly; and he also showed that often the swelling of the feet precedes the ascites.

The extreme rapidity of some cases of alcoholic cirrhosis is very remarkable. Thus Case 3 was dead in nine weeks from the time at which he first felt ill, Case 19 in less than six months, Dr. Carrington's case in less than five months; and then in some cases the duration of the illness is even shorter, for the patient's attention is first attracted to his illness by the ascites. This was so in Case 7. He said he had always been well till he noticed his abdomen enlarging, which was seven weeks before death. The same is true of Case 22, who was well till he observed the abdominal swelling which preceded death by six weeks. In many cases the history is too imperfect to help us with regard to this point, and in others a history of a year or more is distinctly given, but all these rapid cases showed after death extreme cirrhosis, so that we are compelled to assume —for the liver could hardly get to that condition in a few weeks—that sometimes the disease of the liver may remain latent for a long while, then suddenly serious symptoms supervene, ascitic fluid is poured out, the feet swell, and the patient will be dead in a few weeks.

It really appears as though, in many cases of alcoholic

[1] Op. cit.

cirrhosis of the liver, the pathological change in the liver was not in itself of much importance; for not only do we often find an extreme degree of cirrhosis of the liver in persons who have died of something, such as an accident, that can have no connection with the cirrhosis, but patients with alcoholic cirrhosis of the liver are liable to the rather sudden supervention of a series of symptoms, of which the chief are a general feeling of illness, ascites, swelling of the feet, and finally nervous symptoms, as drowsiness and coma. And, further, I think that in many cases the swelling of the feet and the ascites are not, as has been commonly supposed, merely direct mechanical effects of the cirrhosis, but they have some deeper cause of which we know nothing. Lastly, we should not speak of the cure of alcoholic cirrhosis of the liver, for such a phrase is not only pathologically impossible, but the patient is always very liable to succumb to the above symptoms, and to any chance illness he may encounter.

### 3. *Cases which were regarded as Cirrhosis of the Liver, but which were tapped oftener than once.*

Among the sources from which I have collected my cases I have found ten (Cases 25—34) which come under this heading. They fall naturally into two groups. Firstly, those in which cirrhosis of the liver was thought during life to be the cause of the ascites, but in which after death no cirrhosis was discovered (in one case there was slight cirrhosis). There are four of these, and in all the ascites was found to be due to chronic peritonitis. Secondly, there are those in which, at the post-mortem examination, well-marked cirrhosis was found, and inasmuch as they had all survived a second tapping, they at first sight refute my opinion as to the grave significance of the onset of this symptom, but I think I shall be able to show that in each it is possible that the ascites may have been due to some other cause than cirrhosis.

We will now examine the first group.

They are all instances of chronic peritonitis or perihepatitis. Only one (No. 34) of these is an example of malignant disease, and that is the last. I have recorded it because when the patient was first in the hospital under my

care we thought that he was suffering from cirrhosis of the liver. He had considerable ascites, but he improved under treatment by rest in bed, diuretics, and restraining the amount of fluid he drank. He was discharged from the hospital, and had he not returned to Guy's he would almost certainly have been regarded as a case of cirrhosis with ascites which recovered or at least improved very much under treatment, but fortunately for pathology and clinical medicine he did come back. He died about twenty months after he first came in, and it was found that he was suffering from colloid disease of the peritoneum, and that he had no cirrhosis.

Case 25 was that of a man who gave a history of having drunk large quantities of beer. He was admitted for ascites. He was tapped, but the fluid collected again, and before he died paracentesis was performed three times, and during life it appeared probable that he had cirrhosis of the liver. It was found after death that he was really suffering from chronic peritonitis and perihepatitis, and that he had no cirrhosis of the liver. Case 26 closely resembled the last. He was tapped three times and a diagnosis of cirrhosis of the liver was made, but he also was found not to have this disease, but to be suffering from chronic peritonitis and perihepatitis. Case 27 is very similar. When she was first in the hospital she had ascites, was tapped, and the liver was felt three inches below the ribs. A diagnosis of cirrhosis was made. She improved so much that she was discharged, and would no doubt, if she had not come back to Guy's Hospital, have been regarded as a case of cirrhosis cured by treatment; she, however, returned, was tapped again, and died. There was found severe perihepatitis, general peritonitis, and the liver was but slightly cirrhotic. Now in none of these four (Nos. 25, 26, 27, 34) cases, all of which were regarded as uncomplicated alcoholic cirrhosis of the liver, and all of which were tapped oftener than once, was the ascites due to cirrhosis, for in three of them it was entirely absent, and in the remaining one it was trifling; but in all four there was chronic peritonitis, which was undoubtedly the cause of the ascites. As these four cases have all occurred in one hospital in the short

space of five years, they strongly support my contention that if a case which is thought to be cirrhosis of the liver survives the first paracentesis there is a very strong probability that the diagnosis of cirrhosis is wrong, and that the patient is really suffering from chronic peritonitis.

These cases naturally pave the way to the second group, viz. Nos. 28, 29, 30, 31, 32, and 33. In each of these there was undoubted cirrhosis of the liver, each was tapped oftener than once, and therefore at first sight they appear to tell against the view that I have been urging; but it is particularly to be observed that in each chronic peritonitis or perihepatitis was at the autopsy found to be present. It is of course well known that a slight thickening of the capsule of the liver may be met with in cases of cirrhosis of the liver, but I do not think that this will explain the perihepatitis met with in these cases. Certainly it will not in cases in which the capsule was much thickened; and even in those, such as Case 28, in which the thickening of the hepatic capsule was slight, there was some chronic peritonitis, if we may judge by the fact that that membrane was thickened; so that even if we do not regard the perihepatitis as part of the chronic peritonitis, this latter condition may nevertheless account for the frequent collection of ascitic fluid. Now we have seen that uncomplicated cirrhosis of the liver often does not cause ascites at all; and that if it does the ascites is usually a late development, so that the patient does not long survive it. On the other hand, it is known that perihepatitis and chronic peritonitis are a common cause of ascites, and that, as I shall presently quote cases to prove, the patient may long survive the first appearance of ascites and may be tapped many times. Therefore it seems that in the cases under consideration it is more likely that the perihepatitis or the chronic peritonitis was the cause of the ascites than that it was due to cirrhosis of the liver, so that these cases probably ought not to be regarded as instances of ascites due to cirrhosis of the liver which have survived more than one tapping, but rather as examples of cirrhosis of the liver (perhaps latent) accompanied by perihepatitis or chronic peritonitis causing ascites. This is rendered more likely by the

fact which I have already pointed out, that alcoholism and syphilis are common accompaniments of both conditions. These cases, therefore, do not, I think, affect my original contention, that by the time that uncomplicated alcoholic cirrhosis of the liver has caused ascites the patient is so near his end that it is very improbable that he will live long enough to require paracentesis oftener than once.

Then, too, it must be remembered that it is quite possible that in some of these cases the cirrhosis was not alcoholic at all, but may have been a secondary result of the perihepatitis. This was Dr. Murchison's opinion of Case 33, and the account of the autopsy of Case 29 states that "bands of fibrous tissue seemed to run in from the capsule." Case 32 is probably not of much value either way, for while the "thickened omentum" is evidence of chronic peritonitis, it is quite possible that the first tapping set up recent peritonitis, which was the cause of the reaccumulation of the fluid.

It is, of course, not necessary for me to show that, in cases of cirrhosis of the liver in which death followed the first paracentesis, no chronic peritonitis was present; for cirrhosis of the liver is a dangerous malady, which may cause rapid death whether or not chronic peritonitis is present.

I need not at any length support my contention that perihepatitis does not, when sufficiently severe to cause ascites, exist apart from general chronic peritonitis, for I have already published cases[1] which, I think, prove the point. I may perhaps, however, be allowed to quote the results at which I arrived.

I took forty cases quite indiscriminately. Eighteen of them were examples of partial, and twenty-two of complete perihepatitis.

Of the eighteen partial cases six were examples of peritonitis due to either tubercle or cancer, and the thickening of the capsule of the liver appeared to be merely part of the general peritonitis, and therefore we need not consider them any more. The point to which I would very particularly direct attention is, that out of the remaining

[1] 'Clin. Soc. Trans.,' vol. xxi, p. 219.

twelve only one is stated to have peritonitis, and of the remaining eleven eight are distinctly stated not to have had peritonitis, and no mention of the peritoneum is made in the remaining three. Nine of the twelve had capsulitis of the spleen, thus bearing out Wilks and Moxon's statement of the great frequency of the association. In ten out of the twelve there was lung or heart disease, and thus there was a strong probability of backward pressure; of the remaining two, one was a case of cirrhosis, the other was a case of renal calculus.

Now let us examine the twenty-two cases of universal perihepatitis. In the first place, in only two of them is it stated there was no peritonitis. In seventeen of them it is distinctly stated that there was peritonitis, and in the remaining three no mention is made of the peritoneum. It would take too much time to detail the description of the peritonitis. It was always chronic, and was never due to tubercle or growth. I copy one description taken at random:—"The peritoneum was converted into a thick tough layer, matting all the intestines together, and covering them with a thick fibrous apron. The omentum was all matted up into a thick lump, two inches below the stomach." In six cases there was a history of alcoholic excess, and it is particularly noteworthy that in none of these cases was there any marked cirrhosis; in fact, in many cases it is distinctly stated that the liver is soft.

There was never any mention of dilatation of the portal vein.

The number of tappings varied from one or two up to eight or nine, and one case was tapped thirty-five times.

That the cause of the ascites is the general chronic peritonitis is indicated by the fact that it is always present when there is ascites. On the other hand, there is no peritoneal effusion in the rare cases of perihepatitis without peritonitis; and that the cause of the ascites cannot be due to pressure on the portal vein or liver as a whole is shown by the fact that the vein was never described as dilated; it was not dilated or nipped by the new fibrous tissue in a case I dissected, and there is never any jaundice in cases of uncomplicated perihepatitis.

To sum up: I have tried to prove that—

1. The supervention of ascites in uncomplicated cirrhosis of the liver means that the end is near.

2. That if a case of ascites with cirrhosis of the liver lives long enough to require a second paracentesis, it is in the highest degree probable that either the diagnosis is incorrect, or at any rate some other cause than cirrhosis exists to explain the ascites.

3. Such cases nearly always turn out to be examples of chronic peritonitis with perihepatitis, which is not a special disease, but merely a part of chronic peritonitis.

4. Cirrhosis of the liver is often latent, but its chief danger consists in the fact that patients suffering from it are liable to the more or less sudden supervention of symptoms which rapidly increase in severity, and usually soon end in death. The chief are a general feeling of illness, ascites, swelling of the feet, and drowsiness.

5. The ascites and the swelling of the feet are often not direct mechanical effects of the cirrhosis, and the swelling of the feet is often not due to the ascites.

## Appendix of Cases.

Case 1. *Cirrhosis with ascites fatal before tapping was necessary.*—Isaac B—, æt. 68, admitted under Dr. Pye-Smith, July 8th, 1891; clinical clerk, Mr. J. W. Taylor. The only important fact discoverable was that the abdomen contained fluid; the patient gradually sank and died.

*At the autopsy* the liver was found to be typically cirrhotic. There were two pints of fluid in the abdominal cavity, and there was thrombosis of the portal vein.

Case 2. *Cirrhosis with ascites; death before tapping was necessary.*—Henry C—, æt. 53, admitted under Dr. Goodhart, February 28th, 1891; clinical clerk, Mr. F. G. Stern. The patient has always been a heavy drinker. He first began to feel ill six months ago, suffering much from indigestion. He has noticed blood in his motions. Four weeks ago the abdomen began to swell.

*On admission* there was obvious ascites, a slight trace of albumen, and râles at the bases of the lungs. He became delirious, and died March 11th, 1891.

*At the autopsy* there was found typical cirrhosis of the liver, and three and a half pints of ascitic fluid in the abdominal cavity.

Case 3. *Cirrhosis with ascites; death before tapping was necessary.*—John O—, æt. 53, admitted into Philip Ward under Dr. Pye-Smith, July 15th, 1890; clinical clerk, Mr. W. A. Haslam. The patient came in for jaundice and dropsy. He has drunk a good deal of gin. He first became ill seven weeks ago. Œdema of his feet began a few days ago. The liver could be felt to be enlarged, hard, and irregular. He had albuminuria. There were rhonchi in both lungs. The abdomen contained fluid. The patient gradually became drowsy, had wandering delirium, sank, and died July 31st, 1890.

*At the autopsy* the liver was found to be cirrhotic. The rest of the organs were healthy.

Case 4. *Cirrhosis with ascites; death before tapping was necessary.*—Arabella Y—, æt. 46, admitted under Dr. Pavy, April 21st, 1888; clinical clerk, Mr. H. P. O. Manning. She has had rheumatic fever, and has drunk a good deal of alcohol; she has been jaundiced for some time, and recently the abdomen has begun to swell. She has had much abdominal pain, and the day before admission she was restless and had delusions.

*On admission* she was found to be jaundiced, and had several petechiæ under the skin. The abdomen contained much fluid, measuring $35\frac{1}{2}$ inches in circumference at the umbilicus. The liver could be felt to be enlarged. The urine contained bile. She was delirious at night, but, however, became quieter, and ultimately apathetic, lying quite still without noticing anything which was going on around her. She gradually became weaker, and died on May 7th, 1888.

*At the autopsy* there was found cirrhosis of the liver and a quantity of ascitic fluid in the abdomen.

Case 5. *Cirrhosis with ascites fatal before tapping was necessary.*—James R—, æt. 35, a cook, admitted into Stephen Ward under Dr. Washbourn, 1st August, 1891; clinical clerk, Mr. A. H. Card. Until quite lately he has always drunk six pints of beer a day. Two years ago had influenza badly; he was then slightly jaundiced. First noticed his feet swollen two days ago; the next day the penis was swollen, and he had considerable abdominal pain.

*On admission* the legs, scrotum, and penis are very œdematous; there is a considerable amount of fluid in the abdomen; there is some jaundice; the liver could not be felt; the respiratory and circulatory systems were normal; there was a trace of albumen. Shortly after admission he had a considerable hæmorrhage into the scrotum. He gradually developed signs of bronchitis, the swelling of the legs increased, the patient became weaker and weaker, and he died on September 4th.

*Autopsy.*—There were eight pints of fluid in the abdominal cavity. The liver weighed 65 oz., and was much cirrhosed. The spleen was hard. The walls of the stomach were thickened and chronically congested. The kidneys were slightly congested, but otherwise normal. The heart weighed 13 oz., but there was no valvular disease. The lungs were congested. The aorta was atheromatous.

Case 6. *Cirrhosis; ascites; death before tapping was necessary* (Dr. F. Taylor, 'Guy's Hosp. Rep.,' vol. xxxvi, p. 11).—Alfred H—, æt. 18, had for four or five weeks before admission noticed that his abdomen was swelling.

*On admission* ascites was found to be present. He was treated with Mistura Copaibæ Resinæ. Ten days after admission he became comatose, and shortly after he died.

*Autopsy.*—There was well-marked cirrhosis of the liver.

Case 7. *Cirrhosis; ascites; death before tapping was necessary* (Dr. F. Taylor, 'Guy's Hosp. Rep.,' vol. xxxvi, p. 16).—A man, æt. 63, was admitted into Guy's Hospital. He said his abdomen had only been enlarging for two weeks. He was always well till then. It measured 40

inches in its greatest circumference, and evidently contained fluid. He was treated with purgatives and Mistura Copaibæ Resinæ, but he died comatose five weeks after admission.

*Autopsy.*—The liver was extremely cirrhosed.

Case 8. *Cirrhosis; ascites; death before tapping was necessary* (Dr. F. Taylor, 'Guy's Hosp. Rep.,' vol. xxxvi, p. 9).—A woman, æt. 49, was admitted with ascites, which was stated to have commenced eighteen months ago. Three weeks ago the legs swelled.

*On admission* there was ascites, the abdomen measuring 45½ inches in circumference. For the first month she was treated with copaiba resin, and the ascites diminished, but six weeks after admission she became comatose and died.

*Autopsy.*—The liver was extremely cirrhotic. It weighed 47 oz.

Case 9. *Cirrhosis; ascites; death before tapping was necessary* (Murchison, 'Diseases of the Liver,' Case civ).—A man æt. 52, presenting all the signs of cirrhosis, very considerable ascites, and œdema of the lower extremities and scrotum. He gradually grew weaker and died.

*Autopsy.*—Liver weighed 43¾ oz. It was typically cirrhotic. Peritoneum contained several gallons of fluid, but it was otherwise normal. The lungs were œdematous.

Case 10. *Cirrhosis; ascites; death before tapping was necessary* (Murchison, 'Diseases of Liver,' Case cix).—A woman, æt. 32, a year ago began to suffer from abdominal pain and dyspepsia. Four weeks before admission it was discovered that she had ascites.

*On admission* there was considerable œdema of the lower extremities and obvious ascites, which gradually increased. She became delirious and restless, and died eight weeks after admission.

*Autopsy.*—Abdomen contained many pints of clear fluid. The liver was typically cirrhotic; it weighed 36½ oz.

Case 11. *Cirrhosis; ascites; death after one tapping.*—James W—, æt. 59, admitted under Dr. F. Taylor, October 1st, 1892; clinical clerk, Mr. G. T. Birdwood. Has been a

heavy drinker for the last two or three years, getting drunk two or three times a week. Ten days ago the abdomen began to swell, and as it increased he came up to the hospital.

*On admission* he is jaundiced and his feet are swollen. The abdomen contains a large amount of fluid, the liver cannot be felt. The heart and the urine are healthy. There are some râles at the bases of the lungs. Paracentesis abdominis was performed soon after admission, and five pints of fluid were withdrawn.

October 2nd.—During yesterday and to-day the patient has been very restless, and at times delirious.

3rd.—Is getting drowsy. The patient became comatose and died this evening.

*Autopsy.*—A large cirrhotic liver; all the other organs healthy.

Case 12. *Cirrhosis with ascites; death after one tapping.*—Albert P—, æt. 42, admitted December 15th, 1891, under Dr. Goodhart; clinical clerk, Mr. R. Ingram. He came in for swelling of the abdomen and legs. It is said that jaundice was first noticed two years ago. The abdomen and legs began to swell fourteen days ago.

*On admission* there was well-marked jaundice. The abdomen was very tense; the heart and lungs appeared healthy.

December 17th.—Paracentesis abdominis was performed, and twelve pints of fluid were drawn off. The patient became drowsy, and he gradually sank and died on December 24th.

*At the autopsy* the liver, which weighed 60 oz., was typically cirrhotic. There was slight capsulitis of the spleen. Otherwise the whole of the body was normal.

Case 13. *Cirrhosis; ascites; tapped once; fatal a few days after.*—John H—, æt. 59, admitted into John Ward, under Dr. Hale White, April 25th, 1890; clinical clerk, Mr. J. H. Bryant. Patient is a commercial traveller. He first noticed that his abdomen began to swell three weeks ago. The swelling increased till admission, when the abdomen measured 52 inches. He was almost pulseless and very collapsed,

and was therefore tapped at once, 23½ pints being drawn off. His feet were much swollen. Breathing was very difficult. There were rhonchi all over the chest. The tapping relieved him somewhat, but he became very drowsy, and gradually sank, and died on May 4th. He had no albuminuria.

*At the autopsy* the heart was fatty; the mitral and tricuspid valves were a little thick. There were hæmorrhagic erosions in the stomach. The liver was contracted and cirrhotic. There was a little thickening of the capsule, but not much, and it was clearly secondary to the cirrhosis. The kidneys were healthy. No peritonitis.

Case 14. *Cirrhosis with ascites; tapped once; fatal shortly after.*—George B—, æt. 37, admitted under Dr. F. Taylor, May 1st, 1889; clinical clerk, Mr. F. Wellford. He came in for swelling of the legs and abdomen. He occasionally drinks to excess. About seven or eight weeks ago he first noticed swelling of the feet and abdomen.

*On admission* legs and abdomen very œdematous. Girth round the umbilicus 42 inches. There is much ascites. The liver could not be felt. The heart and lungs appeared healthy. There was a trace of albumen.

May 7th.—The abdomen was tapped, and 19½ pints were withdrawn.

9th.—The fluid is beginning to re-collect; the patient is drowsy. From this date the cerebral symptoms increased, he became more drowsy and unconscious, passing everything under him. He died May 19th.

*At the autopsy* the liver was typically cirrhotic; it weighed 65 oz. All the other organs were healthy.

Case 15. *Cirrhosis with ascites; fatal after one tapping.*—Robert F. N—, æt. 64, admitted under Dr. Pavy, January 23rd, 1889; clinical clerk, Mr. A. E. Tebb. He came in for general œdema; he was unable to give any account of himself, as he was in a dull and apathetic condition, but it is said that the abdomen has been swelling for four months. He has drunk much whiskey.

*On admission* there is great swelling of the abdomen, legs, and scrotum. The liver cannot be made out. The

pulse is very small. The heart and lungs appear healthy. There is no albuminuria. Three Southey's tubes were inserted, one in each leg and one in the abdomen, and several pints of fluid drained away. No treatment seemed to do any good, and he gradually became more and more drowsy, and finally sank and died February 4th, 1889.

*At the autopsy* the liver was typically cirrhotic. The other organs were healthy. There was an ante-mortem clot in the portal vein.

Case 16. *Cirrhosis with ascites; fatal after first tapping.* —Sarah S—, æt. 48, admitted under Dr. Pye-Smith, 25th January, 1888; clinical clerk, Mr. F. Vicars. She has suffered from dyspepsia and from piles for some time past. Two months ago she first noticed the ankles and the abdomen to be swollen. The swelling has increased lately.

*On admission* she is slightly jaundiced; the tongue is furred. The abdomen is much distended, and measures 34¾ inches in circumference at the umbilicus. The liver is felt to be about an inch and a half below the ribs. The urine contains bile.

February 20th.—She has been getting weaker since admission. Paracentesis of the abdomen was performed this afternoon; six and a half pints of fluid were withdrawn. She continued to sink, and died 24th February.

*At the autopsy* the liver was typically cirrhotic. It weighed 50 oz. The rest of the organs were healthy.

Case 17. *Cirrhosis with ascites; death a few days after the first tapping.*—Thos. M—, æt. 52, admitted May 15th, 1890, into John Ward under Dr. Pitt; clinical clerk, Mr. A. H. Meadows. He has been a moderate drinker. Four months ago he first noticed his abdomen began to swell.

*On admission* he had considerable ascites, slight jaundice, and râles in the lungs. Eight pints of fluid were drawn off from the abdomen. He gradually sank, and died May 22nd, 1890.

*At the autopsy* the liver was found to be cirrhotic, the mitral valve was thickened, and the mesenteric glands were caseous.

Case 18. *Probable cirrhosis with ascites; death after one tapping.*—Bridget D—, æt. 52, admitted under Dr. Pye-Smith, 14th July, 1891; clinical clerk, Mr. F. Bearblock. She has drunk heavily. She has not been feeling well for six months. Three weeks ago she first noticed that the abdomen was swelling.

*On admission* there was considerable ascites, but no jaundice. The heart, lungs, and urine appeared normal.

July 20th, 1891.—The abdomen was tapped, and eleven and a half pints were drawn off. The liver could then be felt to be enlarged two inches below the ribs.

August 9th.—The patient left the hospital.

I wrote to find out what became of her, and I heard she died the same month.

Case 19. *Probable cirrhosis with ascites; fatal after one tapping.*—Mary Anne H—, æt. 45, admitted under Dr. Shaw, 22nd May, 1891; clinical clerk, Mr. W. Carling. She has drunk a good deal of alcohol. Three months ago she began to have vomiting and sickness. Five weeks ago she first noticed that she was suffering from piles, and then she observed that her abdomen was beginning to swell.

*On admission* she was jaundiced. The abdomen was much distended with fluid. The liver could not be felt. The urine was bile-stained. She suffered from indigestion. There was no albumen in the urine, and the heart and lungs appeared healthy.

June 15th.—Twenty pints of fluid were withdrawn from the abdomen. It soon began to collect again, and on 28th June she went out at her own wish.

I wrote to her friends to find out what had become of her, and I heard that she died a month after leaving Guy's.

Case 20. *Cirrhosis; ascites; death after one tapping* (Dr. F. Taylor, 'Guy's Hosp. Rep.,' vol. xxxvi, p. 10).—A man æt. 58. He had three gallons of fluid removed from the abdomen on October 4th, 1874, but on his admission on October 23rd there was considerable ascites. The patient got weaker; he became delirious and unconscious, and died October 31st.

*Autopsy.*—The liver was very cirrhotic. The left lung was compressed by fluid in the left pleural cavity.

Case 21. *Cirrhosis; ascites; death after one tapping* (Dr. F. Taylor, 'Guy's Hosp. Rep.,' vol. xxxvi, p. 19).—A man, æt. 46, was admitted September 3rd, 1874. He had been unwell nine months, and had noticed swelling of the abdomen four months.

*On admission* it measured 49 inches in circumference. On September 21st three and a half gallons of ascitic fluid were removed by paracentesis. Restlessness, delirium, and coma followed, and he died September 25th.

*Autopsy.*—Liver much cirrhosed and very fatty.

Case 22. *Cirrhosis; ascites; paracentesis once; death* (Frerichs' 'Diseases of the Liver,' Syd. Soc. trans., vol. ii, obs. vi, p. 55).—A man, æt. 66, was admitted into the hospital, having suffered from ascites for five weeks without œdema of the feet. He was quite well till this. Paracentesis abdominis withdrew ten quarts of clear fluid, but the patient died a few days afterwards.

*Autopsy.*—The liver was a typical case of cirrhosis; it was in many places adherent to the surrounding parts.

Case 23. *Cirrhosis; ascites; paracentesis once; death* (Frerichs' 'Diseases of the Liver,' Syd. Soc. trans., vol. ii, obs. ix, p. 63).—A woman æt. 28. She had been in the habit of drinking brandy; four weeks before admission into the hospital her appetite failed; the abdomen had become swollen and her skin yellow. Three weeks before admission the legs began to swell. Three weeks after admission paracentesis abdominis was performed, a large quantity of fluid was withdrawn, but she died a fortnight afterwards.

*At the autopsy* the liver was found to be typically cirrhotic.

Case 24. *Cirrhosis; ascites; tapped once; death* (Murchison, 'Diseases of Liver,' Case cx).—A man æt. 47. Three weeks before admission the abdomen began to swell.

*On admission* much ascites and swelling of the legs. Paracentesis was performed, and sixteen pints of fluid were

evacuated. Four days afterwards, as the fluid was still draining away, an attempt was made to close the opening, but the patient died the next day.

*Autopsy.*—There was recent peritonitis in the neighbourhood of the puncture. The liver weighed 68 oz., and was a typical example of cirrhosis.

Case 25.—*Chronic peritonitis and perihepatitis resembling cirrhosis; tapped three times; death.*—Robert B—, æt. 56, admitted under Dr. Pye-Smith, September 28th, 1892; clinical clerks, Messrs. F. Cowan and L. A. Parry. He came in for swelling of the abdomen and legs, and pain across the epigastric region. Has been accustomed for thirty years to drink six pints of beer a day; latterly he has drunk whiskey instead. No history of syphilis. Present illness began in July with a difficulty in passing his water. Soon after he noticed swelling of the ankles, and soon after this the abdomen began to enlarge.

*On admission* the abdomen obviously contains a large amount of fluid. The legs are œdematous below the knee. The liver cannot be made out. The apex-beat is half an inch outside the nipple. The urine contains some albumen. Some of those who saw him considered that he had cirrhosis of the liver.

29th.—Paracentesis abdominis; 14½ pints withdrawn. After this the albuminuria disappeared.

October 12th.—Paracentesis to 14 pints, as the fluid had re-collected.

28th.—Abdomen again tapped, and 58 fl. oz. drawn off. During the whole of his stay in the hospital the patient got worse; he developed bronchitis and pleurisy, had diarrhœa from time to time, and gradually sank and died on November 21st, 1892.

At the *autopsy* there was found universal chronic peritonitis together with perihepatitis. The liver was not cirrhotic.

Case 26.—*Chronic peritonitis and perihepatitis resembling cirrhosis; tapped three times; death.*—Richard C—, æt. 46, admitted under Dr. Pye-Smith, May 28th, 1892;

clinical clerks, Messrs. L. Slater and F. Cowan. He came in for pain and swelling of the abdomen. He says he drank, but gave it up nine years ago; he is, however, very reticent on the subject. There is no history of syphilis. He has been in the Army, but left nineteen years ago; since then he has done light work in a blacksmith's shop.

*History of present illness.*—A week ago he noticed that the abdomen began to swell. He has also suffered since then from abdominal pain. He has not been sick, nor has he had piles.

*On admission.*—The venules of the cheeks, forehead, and nose are conspicuous, owing to their distension. The tongue is furred, the appetite is good, the bowels are regular. The abdomen is distended, the walls are tense, and a thrill can be felt. The flanks are dull, but become tympanitic when the patient is turned over. The liver cannot be felt on account of the tension of the abdomen. The sounds of the heart are healthy. The lungs are probably slightly emphysematous. The urine contains no albumen. The ankles and lower part of the legs are swollen and pit on pressure. The patient was considered to be suffering from cirrhosis of the liver.

June 4th.—As the abdominal distension has been increasing paracentesis of the abdomen was performed, and ten pints of straw-coloured fluid were drawn off.

14th.—The fluid has evidently re-collected in the abdomen, consequently it was again tapped, and 9½ pints of straw-coloured fluid were drawn off.

16th.—During the last month the patient has suffered much from abdominal pain, which has sometimes been very severe. At times he has been very drowsy. The fluid has re-collected, and to-day the abdomen was tapped. Nine pints of fluid were drawn off.

July 30th.—A pleuritic rub has been heard in the left chest for some few days past. He complains of abdominal pain.

August 5th.—He has been suffering much from constipation. The tongue is furred and dry. The hands and face are cold, and the patient is lethargic.

17th.—He is wasted and thin about the face. He does

not readily understand when spoken to. The tongue is furred and the bowels are constipated.

31st.—He is weak and cold; he dozes the greater part of the day. The mouth is in a sloughing condition, and the breath is very fœtid. From this time he gradually sank, and died on September 7th.

*Autopsy.*—The abdomen contained 15¾ pints of clear straw-coloured fluid. There was universal chronic peritonitis. The liver weighed 26 oz., it was very small; the capsule was thick and opaque, and it presented a typical example of perihepatitis. The thickened capsule was adherent to the diaphragm in places. There was no evidence of cirrhosis. The stomach was extremely congested. The kidneys weighed 9 oz., the cortex was thin. The capsule of the spleen was thick and dense, the organ weighed 7 oz. There were pleuritic adhesions over the bases of both lungs. The heart weighed 7 oz.

Case 27. *Slight cirrhosis; severe perihepatitis; general peritonitis; ascites; tapped twice; death; original diagnosis, cirrhosis of the liver.*—Rose H—, æt. 39, admitted under Dr. F. Taylor, July 16th, 1891; clinical clerk, Mr. F. J. Coleman. She denies having drunk more than half a pint of beer a day. The only thing discoverable on examination was that she had considerable ascites. Ten pints of fluid were drawn off from the abdomen; the liver was felt to be three inches below the ribs. The diagnosis made was that she had cirrhosis of the liver. She improved, and she was discharged on August 10th, 1891.

Readmitted December 24th, 1891, with extreme ascites. She was at once tapped, and much fluid was drawn off, but she died comatose on December 28th, after having had a fit.

*Autopsy.*—There were old peritoneal adhesions. The liver was covered by a dense white capsule, thickest over the right lobe. The anterior edge of the organ was rounded and recurved. The omentum was thickened and drawn up. The liver was extremely fatty and slightly cirrhotic.

Case 28. *Cirrhosis; some chronic peritonitis and perihepatitis; paracentesis four times; death.*—Sarah B—, æt. 59, admitted into Miriam under Dr. Perry, August 17th,

1892 ; clinical clerk, Mr. R. L. Wason. She was admitted for enormous swelling of the abdomen. Previous and family histories unimportant. Four months ago she first noticed her abdomen began to swell.

*On admission.*—The only noteworthy fact is that the abdomen is enormously distended by fluid. She was not jaundiced, nor did she become so.

August 18th.—Paracentesis abdominis was performed, and 9 pints 2 fl. oz. of fluid came away. The liver was then felt 2½ inches below the costal margin, but the surface of the organ was not particularly rough. No growth could be felt *per rectum* or *per vaginam*.

September 12th.—Southey's tube inserted, and 8 pints 5 fl. oz. of fluid were removed. The edge of the liver could be sharply defined 2 inches below the costal margin in the nipple line.

October 1st.—Twelve pints of fluid were removed by paracentesis abdominis.

17th.—Eleven pints and 4 fl. oz. were removed.

21st.—The patient, who had been getting weaker during the whole of her stay in hospital, died to-day.

*Autopsy.*—No signs of recent peritonitis. No growths in the peritoneum, which was, however, somewhat thickened. The liver was slightly smaller than normal, weighing 41 oz. Its capsule was a little thickened and milky, and presented the usual areolated appearance met with in perihepatitis, but Dr. Perry considered the perihepatitis to be secondary to the cirrhosis. The liver itself was nearly smooth, and on section it was very tough and somewhat granular, but not at all the hobnail type of liver, being more like a liver uniformly infiltrated with fibrous tissue. It was, however, cirrhotic. It is remarkable that microscopically it showed small islets of healthy liver tissue. The gall-bladder was thickened and fibroid. There was capsulitis of the spleen. This case is recorded by Dr. Perry in the 'Pathological Transactions' for 1892–3 on account of its showing membranous pharyngitis not due to any irritant.

Case 29. *Cirrhosis ; ascites ; perihepatitis, many peritoneal adhesions ; paracentesis performed twice ; death* (Dr.

F. Taylor, 'Guy's Hosp. Rep.,' vol. xxxvi, p. 17).—Thomas C—, æt. 45, was admitted under Dr. Moxon on June 9th, 1875. He had had abdominal pain and loss of appetite for about five months, and for five weeks his abdomen had been swelling.

*On admission* it was 42 inches in circumference.

On June 21st paracentesis abdominis was performed, and again on July 14th. In addition the patient was treated with purgatives, with copaiba resin and other diuretics, but he died on September 15th.

*Autopsy.*—There was evidence of recent peritonitis. The parietal layer was covered with ecchymosed blood. The transverse colon was adherent to the greater curvature of the stomach, the adhesions being apparently recent. The liver weighed 48 oz.; capsule thickened, white and adherent to the diaphragm at its posterior part. There was a very great increase of its fibrous tissue, this being especially marked along the course of the vessels and towards the surface of the organ, where bands of fibrous tissue seemed to run in from the capsule.

Case 30. *Cirrhosis; perihepatitis; ascites; paracentesis twice; death* (Dr. F. Taylor, 'Guy's Hosp. Rep.,' vol. xxxvi, p. 21).—Robert R—, æt. 40, was admitted May 7th, 1875. His illness began in November, 1874, with dyspnœa and cough; since then he has had bilious attacks, vomiting, and jaundice. The abdomen has been enlarging sixteen days.

*On admission* it is 37½ inches in circumference.

May 10th.—He was tapped, and six pints of fluid were removed. Four days later he was again tapped, and twelve pints were taken away. Coma supervened, and he died May 21st.

*Autopsy.*—Liver exceedingly congested, with much fibrous tissue evenly distributed. Capsule much thickened; the liver also contained a nodule, which appeared to be syphilitic. Lungs emphysematous, with cheesy nodules scattered through them. The testes contained gummata. Other organs healthy.

Case 31. *Cirrhosis; ascites; perihepatitis; chronic peritonitis; twice tapped; death* (Frerichs' 'Diseases of the

Liver,' Syd. Soc. trans., vol. ii, obs. vii, p. 57).—A woman, æt. 54, was admitted into the hospital, having suffered for three years from pain in the abdomen, constipation, and loss of appetite, while the abdomen has gradually become enlarged to a considerable extent. A fortnight before admission the ascites had increased greatly, and the legs had become swollen. Six days after admission paracentesis withdrew twelve quarts of clear fluid from the abdomen. Four weeks afterwards it was again necessary to perform paracentesis, but the patient gradually sank and died three weeks after this.

*Autopsy.*—The liver was contracted with a nodular surface. There was much chronic peritonitis, which had extended along the retro-peritoneal areolar tissue; and portions of the great omentum, the under surface of the liver, the duodenum, the pyloric end of the stomach, and the right flexure of the colon were all firmly adherent. There was serous effusion in both pleural sacs.

Case 32. *Cirrhosis; ascites; tapped twice; death the day after the second tapping; recent and probably chronic peritonitis* (Murchison, 'Diseases of Liver,' Case cxi).—A boy æt. 9. He had drunk a good deal. About a month before admission the abdomen began to swell. There was obvious ascites. By treatment with iodide of iron, citrate of ammonium, mercurial inunctions to the abdomen, and purgatives the ascites subsided in the course of a month, but it soon returned, and in another month paracentesis was performed, fifteen pints being withdrawn. Twelve days after it was again necessary to perform paracentesis; sixteen pints were taken out, but the boy died next day.

*Autopsy.*—The peritoneum contained a few flakes of lymph; it was greatly injected. Great omentum matted into a mass adherent to the adjacent intestines. Omentum thickened and œdematous. Liver only weighed 21½ oz., and was typically cirrhotic.

Case 33. *Cirrhosis; chronic peritonitis; ascites; tapped four times; death* (Murchison, 'Diseases of Liver,' Case cxv).—

A woman in 1850, when 31 years of age, first noticed a swelling under the right ribs. In 1853 the abdomen began to enlarge. On April 17th, 1854, 356 fl. oz. were withdrawn, and on May 19th, 1854, 400 fl. oz.; on June 16th, 1854, 431 fl. oz., and on July 7th 404 fl. oz.; altogether nearly eighty pints. The fluid did not re-collect, but her teeth began to come out, and in October, 1855, a portion of the lower jaw exfoliated; she also had epistaxis and bleeding from the gums. She had never had syphilis nor taken mercury. She sank and died early in 1856.

*Autopsy.*—The liver weighed only 35 oz., and was described as being in an advanced stage of cirrhosis, but Dr. Murchison considered that the firm adhesions of the liver, omentum, and spleen, the thickened capsule of the spleen, and the obliterated cystic duct all pointed to chronic peritonitis as the cause of the atrophy. The spleen weighed 68½ oz., and examination of the blood showed that the patient had suffered from splenic leucocythæmia.

Case 34. *Colloid disease of peritoneum; first diagnosis cirrhosis; many tappings; death.*—Wm. C—, æt. 48, admitted under Dr. Hale White, February 14th, 1891; clinical clerk, Mr. G. F. Birdwood. The patient came in for swelling of the legs and general languor. There is nothing of importance in the family or personal history. He drinks three pints of beer a day. Five months ago the abdomen began to swell, the patient became languid, and had to give up his work. He began to suffer from sickness after breakfast.

*On admission.*—He looks emaciated, his tongue is clean, the abdomen contains a considerable amount of fluid. The girth of the abdomen is 41 inches. There is no pain or tenderness. The heart, lungs, and urine appear healthy. The feet are swollen. The treatment consisted in giving him diuretics, in keeping the bowels open with purgatives when necessary, and in allowing him very little fluid to drink. Under this treatment he improved, and when he was discharged on April 9th, 1891, the abdomen only measured 39 inches in girth, having therefore decreased three inches. The diagnosis was that he was suffering from cirrhosis of the liver.

He was readmitted under Dr. Goodhart September 23rd, 1891. Since he went out in April his abdomen has gradually filled up again. He did not go to work directly, but walked about at his house, and kept coming up for about two months to the hospital to get some medicine (Mist. Potassæ Acetatis: this contains acetate of potassium gr. xx, tincture of squill ♏ xv, spirit of nitrous ether ♏ xxx, juice of broom ♏ lx, water to 1 fl. oz.).

His legs also began to swell, but the swelling went down when he rested. He went on with his work again in June, and at the beginning of this month he was tapped, and thirteen pints of ascitic fluid were withdrawn. In July he was again tapped, and nine and a half pints were taken away. In August the operation was repeated, and eleven and a quarter pints were taken away. He has been continually sick since he has been out. He has been during this time very moderate in his drink; has taken no beer, and only whiskey three or four times. Has had slight pain at the epigastrium. Has never been jaundiced.

*On admission.*—Patient looks wasted; he says he used to weigh over 12 stone, now he only weighs 10 st. 8 lbs.; he has stigmata on the cheeks. The abdomen contains fluid; its girth at the umbilicus is 39½ inches; the liver cannot be felt. The feet are not swollen. The heart and the urine appear healthy. There is an impaired note, with feeble entry of air, at the bases of both lungs. He was ordered Mistura Copaibæ Resinæ, and purgatives when necessary.

October 4th.—Abdomen more distended; girth 41 inches. A Southey's tube was put in, and eleven pints five ounces were withdrawn. After this the size of the abdomen was 36½ inches.

7th.—Diuretic medicine changed to Mistura Potassæ Acetatis.

9th.—Was ordered ten grains of diuretic pill (contains mercury, squill, and digitalis) three times a day.

20th.—The amount of urine passed since the diuretic pill has been ten ounces a day less than with the other diuretics, so he has been put upon Mist. Potassæ Acetatis again.

27th.—The abdomen measures 40½ inches in circum-

ference, so a Southey's tube was put in; two and a half pints were withdrawn.

November 3rd.—The liver is stated to feel as it might if the patient had perihepatitis; also there is something hard to be felt crossing the abdomen just above the umbilicus.

9th.—Six pints of fluid were withdrawn from the abdomen by means of a Southey's tube. This makes it possible to feel the abdomen more easily, and Dr. Goodhart is inclined to think that the mass above referred to is a thickened and puckered omentum drawn up into the neighbourhood of the colon. There is another distinct lump to be felt in the left side of the abdomen.

December 4th.—The abdomen having refilled, a Southey's tube was put in, and four and a half pints were withdrawn. After this the hard masses were easy to feel, and Dr. Goodhart was of opinion that they were omental tumours.

23rd.—The abdomen having refilled, nine pints of fluid were withdrawn by means of a Southey's tube. After this it was clear that there were several lumps to be felt in the abdomen.

February 10th.—The abdomen is very distended. Paracentesis abdominis removed eleven pints of brown fluid. Several lumps could then be felt in the abdomen; the girth afterwards was 39 inches.

5th.—Patient discharged to-day.

*Readmitted* under Dr. Goodhart July 7th, 1892. The abdomen is very distended. It obviously contains fluid, and several lumps can be felt in it.

July 30th.—As purgatives and diuretics have not diminished the amount of fluid in the abdomen, paracentesis was performed, and 6 pints 14 ounces of clear fluid were withdrawn.

August 11th.—The patient has not been so well lately. The abdomen has filled up again; a mass of growth was felt *per rectum*.

31st.—The abdomen is very tense, and the patient, who has been losing flesh and strength lately, complains of acute pain in it; a Southey's tube, however, only withdrew ℥v of fluid.

September 1st.—Patient died.

*Autopsy.*—Liver weighs 45 oz. It is a little soft; its surface is adherent to the diaphragm. It contains no growth. *Peritoneum:* Both the parietal and visceral layers contain masses of colloid growth varying in thickness. The peritoneal cavity contains 118 fl. oz. of fluid. The mesentery is puckered by growth. *Small intestine:* Just above the ileo-cæcal valve there are a few small ulcers. *Large intestine:* The cæcum and cæcal appendix are extensively ulcerated. In other parts of the small intestine are some masses of growth invading it from without, but they have not ulcerated through the mucous membrane. *Stomach* not ulcerated, but is surrounded by masses of colloid material. Spleen and prostate are both invaded by growths. The bases of the lungs are compressed; there are no secondary nodules in them, the kidneys, or the heart.

# THE PATHOLOGY AND TREATMENT

OF

# PUERPERAL ECLAMPSIA.

BEING A PORTION OF A THESIS FOR THE DEGREE OF M.D. IN THE UNIVERSITY OF CAMBRIDGE.

---

By E. PETRONELL MANBY, M.D., B.C., B.A.

(Communicated by A. L. GALABIN, M.D.)

---

I think I may fairly say that while the greatest attention has been, and still is being paid, to the pathology of puerperal eclampsia, we still know very little about it. With regard to treatment, on the other hand, our knowledge has considerably increased of late years, and from the practitioner's point of view this is the most important branch of the subject. I only, therefore, submit that portion of my thesis which deals with these two questions.

It is first necessary to discuss the causation of the albuminuria which, though not present in all cases of puerperal eclampsia, is undoubtedly present in the vast majority.

Why should albuminuria occur in pregnancy at all?

(1) The first theory put forward was that pressure upon the renal veins by the gravid uterus produced passive congestion of the kidneys. This was Lever's view.

The most important objection to this theory is that albu-

minuria is sometimes present in the early months of pregnancy, before pressure on the renal veins can exist. Bartels, the writer on this subject in 'Ziemssen's Cyclopædia,' maintains at some length that even when the uterus is fully enlarged at the end of pregnancy, pressure on the renal veins is a physical impossibility. Dr. King recently, in the 'Lancet,' argued that in the latter months of pregnancy the renal vessels and ureters may be pressed upon owing to malposition of the fœtal head. He says the proper position of the fœtal head is in the iliac fossa, not on the brim of the pelvis.

(2) The next theory—that of Halbertsma—supposes the uterus to press not on the renal veins, but upon the ureters. In consequence of this the kidneys will have to secrete against a higher pressure. This cause may operate while the uterus is still in the pelvis. Fibroids are known by pressure on the ureter to produce dilatation above the point of pressure; and there is some direct evidence that this pressure by the pregnant uterus is a true cause, for Löhlein found that in eight out of thirty-two fatal cases of puerperal eclampsia dilatation of one or both ureters was discovered post mortem.[1]

An important ground for the conclusion that mechanical pressure in one or both of the above ways is often an element in the case, is the fact that albuminuria and eclampsia are much more common in primiparæ, the tension of whose abdominal walls is greater than in multiparæ.

(3) A third cause suggested is, that increased work is thrown on the kidneys by their having to excrete the waste products from the fœtus and enlarged uterus. If the kidneys are weak and barely equal to the normal work, this additional strain may be enough to upset them; but as puerperal eclampsia is more common in young and previously healthy women, I think it is rather going out of our way to assume a debilitated condition of the kidneys before pregnancy, to explain a condition met with in pregnancy, so that this cause, though possible in a few, is not probable in most cases.

(4) A fourth cause may be the increased arterial tension

[1] Dr. Gifford Nash, of Bedford, has recently recorded a case of this kind.

which Dr. Galabin and others maintain is always present in pregnancy. This will increase any tendency which may exist to exudation or to diapedesis of leucocytes into the kidney tissue.

(5) The last possible cause is one suggested by the late Dr. Tyler Smith, namely, that of a reflex influence starting from the uterus, and disturbing the circulation or secretion of the kidneys, just as the circulation and secretion of the mammary gland always, and of the salivary gland sometimes, are disturbed in pregnancy.

With regard to this relation of albuminuria to eclampsia, I should like to mention the investigation of Dr. Lantos, of Buda-Pesth (quoted in the 'British Medical Journal' for January 12th, 1889), into the question of what percentage of pregnant women suffer from albuminuria.

He found the urine albuminous in 60 per cent. of 600 newly delivered women, 70 per cent. of 268 primiparæ, and 50 per cent. of 332 multiparæ.

He believes that albuminuria occurring in pregnancy is the result of a reflex irritation of the sympathetic and renal nerves, due to the irritation of the uterine nerves incident upon the enlargement, and later to the contraction and retraction of the uterus. In his opinion albuminuria is a physiological condition in pregnancy, and in fact is of diagnostic value.

Eclampsia he regards as an acute peripheral epilepsy, having its origin in the uterus.

I myself think too much attention is always directed to the albumen in the urine, and no note is taken of the quantity of urea which is being excreted by the patient.

Time after time, in looking through hospital reports of cases of puerperal eclampsia, I have endeavoured to obtain some information on this point, but have never succeeded in finding that the clerk, or anyone connected with the case, has thought it at all necessary to inquire about the urea, while the almost constant presence of albumen is always mentioned. The quantity of urine passed, and its specific gravity, are also often not noted in the report.

This point seems also to have struck Dr. Lusk, as he says "unnecessary stress is laid on the presence or absence of

albumen. It is the renal insufficiency, not the albuminuria, which causes uræmia and convulsions."

I am not aware that, with the exception of Spiegelberg, any of the text-books mention the fact that the quantity of urea present in the urine of a patient, prior to the onset of the convulsions, may be much diminished.

Dr. G. E. Herman, of the London Hospital, says, while writing on the subject of puerperal eclampsia in vol. xxix of the 'Transactions of the Obstetrical Society,' "It is of course no new view that the fits of eclampsia are uræmic, *i. e.* due to the deficient excretion of urea; but this view has not been universally admitted, and, so far as I know, it has been supported only by arguments based on our knowledge of kidney disease generally, and not demonstrated by observations on the amount of urea excreted by puerperal eclampsics."

After saying this Dr. Herman relates two very instructive cases, in which he very carefully examined the urine both before and after the fits. He has since ('Lancet,' vol. i, 1890) published five other cases, and in all of these the urea was found to be present in very much smaller quantity than normal just before and just after the onset of the first fits, but after delivery and when the fits had ceased the quantity rapidly increased. The specific gravity before the fits was high, and rapidly fell afterwards. In the process of recovery, first more water passed through the kidneys and the specific gravity of the urine fell, and then the excretion of urea became re-established.

Dr. Herman has also very kindly told me of five additional cases he has recently had. In all of these the urea was less than normal before the fits, and rapidly increased after they ceased.

To quote one of Dr. Herman's cases: "August 19th, on admission, urea only ·7 per cent. August 20th, no fits since 3.10 a.m., and at 3.30 p.m. urea 1 per cent.; 9.30 p.m. urea 2·3 per cent. August 21st, 5 a.m., urea 2·3 per cent." The other cases are similar, though in no other was the percentage of urea quite so low at the beginning.

After reading this interesting paper of Dr. Herman's I determined, in all future cases that I might have, to inquire

into the amount of urea passed; and although at present I have only had two cases, my results agree with Dr. Herman's very closely. In the first case, a specimen of urine passed shortly before the onset was very small in quantity, had a specific gravity of 1022, and contained 1·6 per cent. of urea. Labour was terminated by forceps, and next day the amount of urea passed had increased to 2·55 per cent. The next day it was only 1·55 per cent., but as the patient had passed between 30 and 40 ounces of urine, and the specific gravity from 1022 on the first day had fallen to 1012, the total quantity of urea passed did not fall far short of 300 grains. Next day she passed 92 ounces of water, and although the percentage of urea was only 1 per cent. the total amount passed in the twenty-four hours was 413 grains. Next day the quantity of urea excreted amounted to 516 grains, and although it did not again reach so high a quantity it remained between 300 and 400 grains a day until I lost sight of her.

In the other case I found, in a specimen of urine taken by catheter previous to delivery by forceps, 1·4 per cent. of urea. Next day the percentage rose to 2·2 per cent., the following day to 3·3 per cent.; then the following day was 2·8 per cent., and continued the next two or three days 2·6 per cent.

Here, then, are fourteen cases in which I know that the urine was carefully examined with regard to the excretion of urea, and in every one of them was there a marked diminution in the quantity excreted immediately before, or about, the time of onset of the first convulsion. Future experience only can show if this diminution of urea is a constant occurrence in these cases, though I venture to think that fourteen examples showing a marked diminution are fairly strong presumptive evidence that such is the case.

With regard to the quantity of albumen, it is generally considerable, often occupying a quarter, or a third, of the bulk of the urine, after the application of heat and nitric acid. In some cases indeed the urine becomes absolutely solid on the application of heat.

There are cases, however, of eclampsia—and I shall have more to say on this subject later—in which either there is no

albumen present from first to last, or in which none is present at the onset of the convulsions, but shortly afterwards it appears, at first in small quantity, later on in profusion.

Dr. Herman has ascertained, in his cases of eclampsia, the extent to which the albuminous precipitate was composed of paraglobulin, and serum-albumen, respectively, in order to test the views of Ralfe and Maguire on the clinical importance of this distinction. Out of nine cases in which the amounts were separately ascertained, he found that in five the albumen was nearly all paraglobulin.

In Bright's disease, on the other hand, it is usually ordinary serum-albumen.

In the four remaining cases, however, Dr. Herman only found small quantities of paraglobulin. I have investigated this point in my two above-mentioned cases, but in neither of them was there any appreciable quantity of paraglobulin present, while serum-albumen was present in large quantity. I also find, from a letter of Dr. W. K. Fyffe to the 'Lancet,' that Dr. Champneys, and Dr. Whipham, at St. George's Hospital, recently had a case where this point was investigated; but while serum-albumen was present to the extent of 75 per cent., paraglobulin was present in very minute quantity. Forty-eight hours later, when there was still 12 per cent. of serum-albumen, all trace of paraglobulin had disappeared.

If continued observation on this point should show that, in cases of simple eclampsia, the albumen is always paraglobulin, it may be, as Dr. Herman says, an important addition to our knowledge of the pathology of this disease. For Drs. Ralfe and Maguire state, that the circumstances in which paraglobulin—the most diffusible kind of albumen—is found in the urine are such as to warrant the belief, that its presence is due rather to altered pressure in the vessels, than to changes in the renal cells; and this, together with the preponderating frequency of eclampsia in first pregnancies, and towards the end of pregnancy, would go to show how important a factor increased pressure is in the production of the disease.

For the causation of the eclampsia outbreak many theories

have been proposed, and I will endeavour to arrange them in a more or less tabular manner.

### I. *Cerebro-spinal Congestion as the Cause.*

This theory was generally held by the older British and American obstetricians, though by many it was not adopted to the exclusion of other theories.

Dewees taught that puerperal convulsions were epileptic, apoplectic, or hysterical. He considered a "strong determination of blood to the head" as a premonitory symptom.

Meigs asserted that the disease was caused by long-continued or violent determination of blood to the head, by the rapid revolution of the blood excited by pregnancy or labour, or by too intense perception of the pains of labour.

Hodge held that convulsions in a large proportion of cases arise from a congestion of the blood-vessels of the brain, or from an actual effusion of serum or blood into its substance or cavities.

I think that nowadays it is generally believed that cerebral congestion is the consequence, not the cause, of the eclampsia.

### II. *Acute Cerebral Anæmia the Cause, according to the Traube-Rosenstein Theory.*

For this theory the primary factor is the hydræmic condition of the blood which is an ordinary concomitant of the pregnant state; and of course when there is also albuminuria, the watery condition of the blood is greatly intensified. Accompanying this condition of the blood there is increased arterial tension, which is favoured by the hypertrophy of the heart which is known to occur in pregnancy.

The result of these combined states is a temporary hyperæmia of the brain, which is rapidly succeeded by serous effusion into the cerebral tissues, resulting in pressure on its minute vessels, and consequent anæmia.

Convulsions are the consequence of anæmia of the bulb, and coma of anæmia of the encephalon.

This theory agrees with Kussmaul and Tenner's and Brown-Sequard's ideas on the relation of cerebral anæmia to convulsions. It also would explain how the occurrence of labour should intensify the convulsions, since during the height of the pains the tension of the cerebral arterial system is necessarily greatly increased.

Hecker answers this theory thus:

(1) "The hydræmia which is a necessary condition for the theory is so common in pregnancy, that if it had an important influence, eclampsia ought to be of much more frequent occurrence;" and on the other hand, as Spiegelberg says, this hydræmia is not always present in eclamptic women —they are, indeed, frequently young and vigorous.

(2) "The increased arterial tension may occur in labour, but is not found in pregnancy and the lying-in state."

This is a point at least open to contest, for Dr. Galabin and others maintain that in pregnancy arterial tension is always increased.

(3) "The anæmia and œdema of the brain are much more naturally explained as secondary to the attacks, than regarded as primary, and the cause of the convulsions. It is frequently observed that consciousness—a correlate of an intact brain—is lost to a greater degree the more frequently the attacks occur."

### III. *Cerebro-spinal Anæmia with Congestion of the Meninges.*

The late Dr. Angus McDonald made two careful post-mortem examinations, and found in both of them intense anæmia of the deeper portions of the brain, especially of the basal ganglia and the cord, with congestion of the meninges, and engorgement of the venous sinuses on the inner aspect of the cranium and spinal canal. He found no œdema of the brain. The theory then proposed by him was, that eclampsia is caused by irritation of the vaso-motor centre, in consequence of an anæmic condition of the blood produced by the retention in it of toxic material, this over-stimulation resulting in anæmia of the deeper seated nerve-centres, and consequent convulsions.

I do not think I need say anything here on the subject of convulsions resulting from cerebral anæmia from vaso-motor spasm. My esteemed teacher the late Dr. Moxon, in his Croonian Lecture in 1881, discussed the subject at great length, in his usual masterly style, and decided that the theory was untenable.

### IV. *A Retention in the Blood of some Toxic Agent, and a Poisoning of the Nerve-centres thereby.*[1]

What is this toxic material?

(*a*) *Urea.*—This was the theory of Braun, of Vienna, in 1851. Against this, although the blood of the patient suffering from eclampsia contains an excess of urea, there is no eclampsia in the cholera patient, whose blood is loaded with this substance.

C. Bernard, B. W. Richardson, and others, injected urea into the blood of living animals, and failed to produce the phenomena of puerperal convulsions.

(*b*) *Carbonate of ammonium.*—This was Frerichs' idea, that the urea was decomposed in the blood into carbonate of ammonium. He made some experiments to prove this, and Spiegelberg, some years later, experimented on dogs, and maintained the accuracy of Frerichs' views.

Hammond, of Maryland, made a series of counter experiments showing the Frerichs theory was incorrect, and Spiegelberg now says, that from his later experiments he can "only regard ammonæmia as one of the rarest causes of the convulsions."

(*c*) *The poison is not urea alone, but in addition various so-called extractives—kreatin, kreatinin, &c.*—The salts of potash have also been suggested by some observers; hence the name urinæmic, instead of uræmic, has been proposed.

With regard to this, however, as Fagge says, the term "uræmia" is generally now taken to mean the poisoning of the nerve-centres by materials accumulated in the blood, as the result of defective excretion by the kidneys. But it is still uncertain whether the poisonous action is excited by one substance in particular, or by all of them together.

[1] This view is the one most generally accepted, I think, and the origin of the poison is usually regarded as arising from failure of the kidneys.

(*d*) Almost the most recent suggestion as to the toxic agent in the blood of eclamptics was made by Doléris and Butte. These observers have found soluble toxic ptomaïnes in the blood of eclamptic patients, which they believe may be the cause of the disease, at least in some cases.

They claim that this discovery permits us to put aside the exclusive idea of renal insufficiency from congestion, spasm, compression, old lesions of the kidneys, &c., and the purely nervous theory, and suggests that eclamptic like epileptic attacks do not have a single etiology.

(*e*) A year later, viz. in 1887, M. Blanc discovered in the urine of eclamptic women a micro-organism which, inoculated into rabbits, produced convulsions. Continuing his investigations, he has recently ('Lyon Médical,' May 12th, 1889) found a peculiar slender bacillus, one or two micromillimetres in length, which, inoculated into rabbits, produces convulsive phenomena, and later on acute nephritis. The blood and fluids of the inoculated animals have also the power of producing convulsions in other rabbits.

From these experiments he concludes, that there are some cases of puerperal eclampsia due to an organism which causes infective nephritis, and this appears to generate a convulsive poison.

(*f*) In September, 1890, Drs. Ehrhardt and Favre of Paris, writing in 'Nouvelles Archives d'Obstét. et de Gynéc.,' trace the eclampsia to nephritis set up by the presence of germs in the placenta. In the placenta of nineteen cases out of twenty of albuminuria these observers found white infarcts, and all the patients except two were subject to leucorrhœa and endometritis when not pregnant.

Colonies of bacteria were found in the infarcts, and when some germs taken from the placentæ of patients who had suffered from eclampsia as well as albuminuria were injected into the veins of rabbits and guinea-pigs, parenchymatous nephritis was set up.

Thus the morbid changes in the decidua which cause placental infarcts are induced by the same agency that induces the nephritis of pregnancy. This agency is the presence of germs in the placenta, which germs produce in non-pregnant patients leucorrhœa, and other symptoms of

chronic endometritis. The focus of infection being the uterine mucous membrane, that structure requires attention and treatment.

I am bound to say that this theory, like the one mentioned just before it, hardly commends itself to the unprejudiced observer, and the investigations of these observers must be repeated and amplified before they become widely accepted.

### V. *Reflex Nervous Impulses as the Cause.*

There are three theories which come under this heading. To put them shortly,—

(1) One which attributes the convulsions to cerebro-spinal disturbance from peripheral stimulation, and quite independent of the kidney.

(2) A second, which suggests that the convulsions are uræmic, *i. e.* due to retained waste products; but the renal disturbance which gives rise to this uræmia itself results from some reflex irritation,—vaso-motor spasm of the small renal vessels, with consequent degenerative changes in the kidneys.

(3) The third theory proposes to explain those cases in which there is no albuminuria from first to last, by considering them as acute epileptic attacks, the area of distribution of the sciatic nerve being the epileptogenic zone.

To take this last theory first; Spiegelberg says of the attacks in these cases that they may be grouped in the same category as the epileptic attacks which can be artificially induced.

Further, there are many facts which show that the nerves of the generative organs actually correspond with very important areas of irritability, which may act as so-called epileptogenic zones.

It would then merely be necessary in any case of eclamptiform paroxysms to demonstrate some unusually great irritation in the pelvis, though the possible existence of individual proclivity, or of special irritability, would also need consideration.

He mentions a case of such convulsions in which the source of irritation probably lay in an enormously distended

bladder, after the emptying of which the fits ceased. Of course, too, La Motte's classical case was precisely similar.

Taking next the first theory, that puerperal convulsions are reflex actions excited by cerebro-spinal or medullary irritation of uterine origin, and transmitted through the ganglionic cells in which the reflex nerves terminate, this is traced back by Dr. Thomas Moore Madden to Laurence Joubert, who was Professor of Medicine in the University of Montpellier in the middle of the sixteenth century. He asserted that the cause was irritation, and only by the removal of this cause could the convulsions be arrested.

Tyler Smith was, I think, the earliest English writer to adopt this theory. He said in 1844, "The true puerperal convulsion can only occur when the spinal marrow has been acted upon by an excited condition of an important class of incident nerves, namely, those passing from the uterine organs to the spinal centre, such excitement depending on pregnancy, labour, or the puerperal state. While the spinal marrow remains under the influence of either of these stimuli convulsions may arise from two series of causes,—those acting primarily on the spinal marrow, or 'centric' causes; and secondly, those affecting the extremities of its incident nerves —causes of 'eccentric' or 'peripheral' origin."

Frankenhauser, of Jena, has demonstrated and illustrated a direct connection between the nerves of the uterus and the renal ganglia. He believes that the sudden occurrence of the eclampsic attack following an external source of irritation (as pressure of the fœtal head on the cervix uteri, digital examination, &c.), and from emotional causes, goes to prove that the nervous system, and not the vascular system, is the starting-point of puerperal convulsions.

The latest adherent to this theory is Pajot, who declines to admit the dependence of eclampsia on albuminuria. He believes that the cause is reflex, culminating in a cerebro-spinal centre in close proximity to the disorganised centre which presides over the existence of albuminuria. He thinks albuminuria predisposes to attacks, but does not act as an exciting cause.

The warmest advocate for the second, the uræmic and reflex nervous theory, if I may so call it, is Spiegelberg.

He says, "We must cling to the central idea that the disease is of the nature of uræmic poisoning, due to an inadequate secretory activity of the kidneys—a view which is not only plausible in virtue of its simplicity, but which explains all symptoms and harmonises with all our experience."

When, in women who have not suffered for some time previously from Bright's disease, eclampsia more or less rapidly develops, this observer says it is with great probability caused by an acute affection of the renal vessels, this vaso-motor spasm being caused in a reflex manner by irritation of the uterine nerves.

It is well known that in the worst cases the bladder may be found for a time entirely empty, and in all cases the quantity of urine secreted is exceedingly small.

That in a number of cases the suppression of urine does occur suddenly is well shown by Hofmeier, quoted by Lusk, who reported 104 cases in which he had found the kidney symptoms develop suddenly.

This sudden suspension of urinary secretion can only result from disturbances of the renal secretion. A rapidly developed affection of the vessels would leave no post-mortem changes, and would, in a case of recovery, disappear as quickly as it had come.

If the renal troubles were due to pressure of the gravid uterus on the renal veins and ureters, renal engorgement and other post-mortem changes should be found in all cases; whereas, although some authorities describe such changes, others—Spiegelberg and Cohnheim particularly—have found the organs oftener pale and bloodless.

The precise nature of this change in the vessels is, either that the vessel walls are altered in such a way as to interfere with the process of diffusion—and this view would seem most probable in those cases in which prodromal symptoms are present; or the disturbance might consist in vaso-motor spasm of the renal vessels.

We know from physiology how sensitive the secreting epithelium of the kidneys is to even brief interruptions in the blood-flow; spasm of the arteries will entirely arrest secretion, and if it occur repeatedly will kill the epithelium, and cause the constituents of the urine to be retained in the

blood. A similar influence of the vaso-motor nerves is known to exist in the case of other organs. There are, moreover, other signs which indicate that such an irritation exists in eclampsia, *e. g.* the wide and sluggish pupil at the beginning of the attack, and the spasm of the cutaneous vessels.

It is not improbable that the vaso-motor irritation proceeds from the uterus—indeed, the association of some special irritation of the sensory nerves of the uterus with the convulsions has been frequently and properly pointed out; and if we suppose that the vaso-motor nerves of the kidneys which run in the splanchnic nerves are irritated in a reflex manner by the uterus, we explain the undoubted causal connection between uterine contractions and convulsions.

I find in the English volume of 'Cohnheim's Pathology,' just published, that this great authority, after discussing the other theories of eclampsia, expresses himself of opinion that the true explanation of the whole subject is provided by the "hypothesis of an arterial spasm of the small renal arteries of reflex origin, that in a moment gives rise to the retention of the urinary constituents in the blood."

There is much to be said in favour of these theories of reflex irritation. By them we can answer two of the three objections which have been raised to the theory of uræmia as the cause of eclampsia.

The objections to the uræmic theory are :—

1. That a woman who has suffered for some time previously from Bright's disease not uncommonly escapes puerperal eclampsia.

2. That in many cases of eclampsia the pathological changes found in the kidneys post mortem are very slight, or they may be absent entirely.

3. Eclampsia may occur without albuminuria at all, or this may not set in until after the convulsions.

1. To the first objection we may answer, that in chronic nephritis there may be sufficient healthy renal structure for the work of elimination; it is only when a fresh inflammation takes place, or when the extra strain thrown on these damaged organs is more than they can stand, that renal insufficiency occurs and eclampsia follows; also in chronic

Bright's disease the nerve-centres become in some measure tolerant of the poisoned blood.

2. The reflex vaso-motor theory answers the second objection. As stated just now, the complete and sudden arrest of the urinary secretion, such as certainly does occur in many cases of eclampsia, must depend on some disturbance of the circulation through the kidney, and such a disturbance need not leave any definite traces post mortem. At the same time, though there need not be any actual post-mortem changes, the vaso-motor renal spasm probably can, as Cohnheim says, give rise in a moment to the retention of the urinary constituents in the blood.

This is the only theory that explains the numerous instances in which, as soon as the paroxysms subside and recovery occurs, all the renal symptoms disappear, and rapid and complete restoration to health follows.

3. The first half of the third objection to the uræmic theory, viz. that eclampsia may occur without any albuminuria from first to last—these cases are very rare, but still they do occur—can be explained by regarding such cases as acute peripheral epilepsy with the pelvic and uterine nerves as the epileptogenic zone. This theory is, I think, now being very generally accepted as a reasonable explanation of these cases.

With regard to the cases where there is no albumen at first, but after a few hours albumen appears in the urine and continues for some days, I have never heard any explanation offered.

These are the cases from which Dr. Braxton Hicks argued that the uræmic theory was erroneous, because at the time of the fit there was no nephritis—albuminuria being taken as the only evidence of renal change; and to many other observers it is these cases which seem to have formed a considerable objection to explaining the convulsions as due to the retention of urea in the system, it not having been suggested that this could arise otherwise than by an antecedent nephritis accompanied by albuminuria.

I should like, therefore, to direct particular attention for a short time to these cases, as it seems to me that a possible explanation can be found by considering the physiology of

the kidney. This organ is described as consisting of two portions, a glomerular and a tubular portion ; and the epithelium of these two portions has very different functions. To quote Dr. Michael Foster, "We may for the present conclude that the secretion of urine does consist of two separate and distinct acts—glomerular and tubular secretion. The work of the epithelium of the tubules is largely if not entirely confined to simply picking the urea out of the blood, and pushing it, so to speak, into the lumen of the tubules.

"The work of the epithelium of the glomerular portion, on the other hand, is mostly concerned with the passage of water from the blood, together with some of the soluble diffusible constituents of the blood ; and so long as the epithelium of the glomerular loop is intact and sound, there passes through neither albumen nor globulin."

Next, on the authority of Landois and Stirling, Litten, and Ribbert, we learn that ligature of the renal artery, even when it is obliterated for only two hours, causes necrosis of the epithelium of the uriniferous tubules. If the ligation be continued for a longer time the epithelium of the glomeruli becomes greatly changed, and finally the whole renal tissue dies, unless the ligature be removed.

In order to be quite certain that these statements were accepted in the physiological world I wrote to Dr. Starling, of Guy's Hospital, and he replied, "The observation you quote is quite correct. In many cases if the artery stopped is small the glomerular epithelium does not degenerate at all."

Physiologists are also in the habit of speaking of the existence of a "renal centre" for the control of the nerves of the renal plexus. This centre lies in the floor of the fourth ventricle, in front of the origin of the vagus.

The relation of the blood-supply to, with the secretion of urine by, the kidney, and the vaso-motor arrangements of the kidney, are too well known to be put down at any length here.

Now to attempt to explain by these physiological facts the particular cases of puerperal eclampsia we are discussing.

First, all authorities agree that during pregnancy the irritability of the nerve-centres is greatly increased ; in fact, there is a sort of general hyperæsthesia.

Suppose now that, as a result of some peripheral or emotional disturbance, a stimulus from the renal centre descends along the vaso-motor nerves to the kidneys in the form of vaso-constrictor impulses, what will happen?

In some instances these impulses may produce such spasm of the vessels of the kidneys that total suppression of urine may occur, lasting, according to Spiegelberg and Overbeck, as long as three quarters of an hour. But suppose this reflex contraction be not so intense as to totally stop the secretion of urine, but sufficiently intense to damage in an hour or so the epithelium of the uriniferous—or urea-eliminating—tubules, and so lead to a retention of this substance in the system; we have here the case of the patient previously apparently healthy, *and without albuminuria,* suddenly developing convulsions as a result of this retained morbid material.

Suppose then that this anæmia of the kidneys from vascular spasm goes on for a longer time, what will happen? Simply that the epithelium of the glomeruli will suffer as that of the urinary tubules has already suffered, and then we have albumen transuding. At first only the urea-eliminating epithelium is damaged, later on that of the glomeruli suffers too.

I offer this as a possible explanation, then, of those cases of puerperal eclampsia where the urine tested at the time of or just before the first fit is found to be free from albumen, but after a time this substance appears—in small quantities at first, in greater quantity later.

To complete the theory, one wants to find that in a specimen of *non-albuminous* urine, excreted immediately before the first convulsion, the amount of urea is diminished, and that as albumen appears the amount of urea increases again. This, however, is very difficult to obtain, because these cases occur, as I have repeatedly said, in the apparently healthy individual and quite suddenly, so that there is no idea of examining the urine until after the fit has occurred.

The objections which may be raised to reflex vaso-motor spasm causing uræmia and nephritis are:—

(1) The theory is based largely on physiological knowledge and experiments. Have we any pathological evidence

of vaso-motor spasm causing destructive changes in the tissues of the human body?

In answer I may cite Raynaud's disease as a good example of pathological change from vaso-motor spasm.

Also Cohnheim, discussing the subject, says that it is probable that the albuminuria so often observed after an epileptic attack, in tetanus, and in lead colic, is a consequence of temporary tetanic contraction of the small renal arteries.

(2) The theory assumes an exciting cause to start the reflex spasm. If the pregnant uterus be regarded as this exciting cause, why do not all pregnant women have eclampsia?

Again, will uterine irritation explain post-partum eclampsia?

Also, will uterine irritation explain the fact that a woman may have nine good confinements and have eclampsia with the tenth?

In meeting the objections to this theory, the first thing to insist on is the great increase of nervous irritability induced by pregnancy. This, though present in all, is much more marked in some women than in others. The next point is that 60 per cent. of all eclampsics are primiparæ. I should like here to remark a point I have never seen mentioned in any text-book, that a not small proportion of these are *unmarried* primiparæ. Hence, in addition to the ordinary heightened nervous irritability, there is a large "mental element" present. This is a point I always now inquire into, and the association is undoubted. I find this point has been noticed frequently by Dr. Horrocks, and probably many other people have noticed it too, but as far as I am aware it has not got into the books.

The exciting cause need not arise from the uterus; fright or other emotional disturbance may be the cause. I know of three such cases. They are, however, instances of "centric," not "peripheral" irritation.

Supposing, however, that the exciting cause be the uterus: the fact that all pregnant women do not have eclampsia can, I should imagine, be explained by the knowledge that all women are not of the same temperament;

some are much more excitable than others, and we can imagine that a stimulus from the uterus, which in one woman would be nothing, would be in another quite enough to start the trouble.

Uterine irritation will, I think, also explain post-partum eclampsia, because, as Dr. Tyson in his monograph on 'Puerperal Eclampsia' says, "the sting of the lash does not cease when the whip is laid down;" and because the uterus has been emptied of the main source of irritation, it does not follow that everything will at once settle down and involution take place. The cervix uteri, at any rate, may be in a very bruised and lacerated condition.

(3) The next objection which might be raised, particularly in the case of my theory, which assumes the convulsions to occur in a comparatively short time after the stimulus, is that the amount of urea and of other urinary constituents which is retained in the system from this vascular spasm is insufficient to cause convulsions in a few hours.

In answer to this, and in spite of certain physiological experiments pointing in the opposite direction, I would submit that we are not able to say anything about the dose of urinary constituents necessary to so poison the blood of a human being that convulsions result. The cerebral centres which, in a case of chronic Bright's disease, may tolerate a large dose, may in a fresh young girl, or in a debilitated woman, behave quite differently.

*For my own part, in conclusion,* I think the mistake so many writers have made is that they have tried to draw too hard and fast a line with regard to the pathology of this disease. The clinical history of the cases undoubtedly varies, and why should not the pathology vary also?

I believe that, until we know more on the subject, the nearest we can get to the truth is by assuming that there are at least four different groups of cases of puerperal eclampsia:—

(1) Cases of undoubted chronic Bright's disease, where pregnancy has thrown on the weakened organs a strain they are unable to bear. These are true cases of uræmia.

(2) Cases where there is no evidence of nephritis from first to last—the so-called cases of acute peripheral epilepsy.

(3) By far the largest group, where the convulsions are uræmic from deficient secretory activity of the kidneys, the first change being spasmodic contraction of the small renal vessels, followed by inflammatory changes in the secreting epithelium akin to acute Bright's disease; this vaso-motor spasm being started reflexly, probably in most cases from the irritation of the uterine nerves, but not necessarily so in all cases.

(4) Perhaps a subdivision of class 3, where in a patient apparently previously healthy convulsions suddenly occur, and albuminuria, though not present at first, appears shortly afterwards. Here the convulsions are the result of uræmia from sudden suppression of urine in kidneys previously healthy. Uræmia is the first result, albuminuria the second.

*Treatment.*—Dr. Barnes puts down "four cardinal principles" for our guidance.

1. Moderate and control nervous irritability.
2. Moderate vascular tension.
3. Cut off emotional and peripheral irritants or excitants.
4. Eliminate all complicating morbid conditions.

He goes on to say that treatment should be—(1) Prophylactic. (2) Remedial. (3) Restorative.

Until recently venesection was regarded as the sheet-anchor in the treatment, and blood was frequently removed in large quantities. Lever, for instance, in the 'Guy's Hospital Reports' for 1843, says "moderate bleedings are of no service," and he took 122 oz. of blood from one patient who recovered!

Dr. Phillips, in 1870, was, I think, the, or one of the earliest to raise his voice against the then almost universal teaching of bloodletting in all cases. He related eleven cases which he had treated with chloroform and without bleeding, every one of which recovered.

In 1872 Charpontier, analysing 133 cases of eclampsia observed under Depard, made out a strong case against bleeding. He showed that the mortality was 41 per cent. under a single bleeding, 54 per cent. under repeated bleeding, 18 per cent. under anæsthetics.

Spiegelberg, Barnes Robert and Faucourt, Lusk, Parvin,

Fordyce Barker, and others, all speak of this still as undoubtedly the most powerful resource at command for rapidly and certainly lowering the vascular tension. Spiegelberg says, "Venesection ranks first in true eclampsia; nothing else has such power of restoring to the kidneys, so sensitive to any alteration in the blood-pressure, their functional activity; few have such a sedative influence on the irritated vaso-motor nerves. I therefore advise the practitioner as soon as he sees his patient, after the first fit, to extract at least seven ounces of blood from the basilic vein."

Lovejoy and Allen, too, report in the 'Boston Medical Journal,' 1887, that they did not lose a single case of eclampsia out of the fifteen from whom they abstracted blood.

At Guy's Hospital, since the time of Dr. Phillips, venesection has not found favour, and Dr. Galabin teaches that it should be left as a last resort in cases in which all other means fail to arrest the convulsions, or where it may seem desirable to relieve extreme venous congestion of the lungs.

It seems to me that we have an argument against venesection from physiology. Dr. Foster says, "When an animal with normal blood-pressure is bled from one carotid the pressure in the other carotid sinks so long as the bleeding is going on, and remains depressed for a brief period after the bleeding has ceased. In a short time, however, it regains or nearly regains the normal height. This recovery of blood-pressure after hæmorrhage is witnessed so long as the loss of blood does not amount to more than 3 per cent. of the body weight. Beyond that a large and frequently a sudden dangerous permanent depression is observed."

All authorities who advocate venesection do so on the ground of lowering the general blood-pressure. From Dr. Foster's statement, to do this at all effectually 3 per cent. of the total body weight has to be abstracted in the way of blood.

Take an average woman as weighing 9 stone, *i. e.* 126 pounds; 3 per cent. of this is roughly 4 pounds. Hence to make any real impression on the blood-pressure 64 ounces by weight of blood must be abstracted. Lever, it is true, advised copious venesection, and, as above stated, once bled a patient 122 ounces; but 17 ounces is about the utmost

advised by the authorities in favour of bleeding, and Lever did not take all his 122 ounces at once, but on two or three occasions during a period of seventeen hours. He would be a bold man, I think, who would take at one time, or within a very short space of time, an amount of blood equal to 3 per cent. of the body weight.

On these grounds it seems to me that venesection as practised for the purpose of lowering the general blood-pressure can be of little or no good to the patient, supposing her to be a strong and previously healthy woman; and if she be anæmic, as many women during pregnancy are, the loss of blood must be actually harmful.

The statements I have quoted from physiology refer to the quantity of blood necessary to be removed from an animal with normal blood-pressure. It is possible that in the case of the puerperal eclampsic, where the blood-pressure is above the normal, the abstraction of a small quantity of blood may lower the pressure. I do not know.

There is no doubt that venesection may in a few cases be an exceedingly powerful agent for good—in relieving an engorged right side of the heart, for instance; but here no suggestion of reducing the general blood-pressure arises, it is simply a rapid means of relieving dangerous venous congestion.

A certain though not particularly rapid method of lowering the arterial tension and relieving congestion is by means of purgatives—elaterium, calomel, croton oil, and salines. Of these croton oil is particularly valuable because it can be given if the patient be in an unconscious condition by simply placing one or two drops on the tongue. Pulv. Jalapæ Co. in drachm doses is an exceedingly efficient purgative in this disorder, though its action is not so rapid or so effectual as croton oil.

Lever used to give his cases tartar emetic in large doses, and he speaks of it as a very important adjunct to bleeding.

The great indication, however, in the management of eclampsia is the controlling of convulsive action by means of *sedatives*. Foremost amongst them is the *administration of chloroform*. There is a general consensus of opinion now in favour of chloroform inhalation, and I do not think there are

many or any practitioners nowadays who, whatever their other treatment may be, do not give some of this anæsthetic with a view to control an actually present convulsion.

Spiegelberg says that having lowered the blood-pressure by venesection, chloroform narcosis should not be induced too soon, as the blood-pressure may be further reduced and death occur.

As Dr. Barnes says, "chloroform is both prophylactic and remedial. Used during the convulsion, it certainly shortens it. By watching for premonitory twitchings and then giving it, the fit may be averted, or certainly mitigated."

Chloroform may be administered continuously for many hours, and so time is gained for the completion of labour, but I think the above-mentioned method of giving an additional quantity when there are indications of an approaching convulsion is a better way than keeping up a continuous narcosis.

*Chloral*, too, has a very beneficial effect in many cases of eclampsia, by moderating nervous and vascular tension and excitement. It may be given alone, but I think the combination with bromide of potassium is a distinct advantage; twenty grains of chloral with thirty grains of bromide may be given, and repeated at intervals of from four to six hours. If the patient cannot swallow, these drugs can be administered with an equally good effect by the bowel. Dr. Playfair speaks of giving chloral hypodermically—six grains to be dissolved in a drachm of water and injected under the skin. Dr. Fordyce Barker is opposed to the use of chloral, which he thinks excites instead of lessens reflex irritability.

*Morphia* has also been much extolled by some authorities—Barker, Hecker, and Clarke of Oswego. Clarke gives one and a half grains hypodermically at once; this dose he repeats in two hours if another paroxysm occurs, and if the patient be in labour another dose at the end of eight hours.

I have only seen morphia given in one case of eclampsia, and certainly it seemed then to be most effectual, no more fits occurring.

Dr. Bowstead, of High Wycombe, in 1869 reported two successful cases treated by morphia, and quite recently Dr. Herman reported five cases at the Obstetrical Society, four

of which recovered, and all were treated thus. Of course morphia is open to theoretical objections in this as in other renal affections, but as physicians are now treating uræmia by morphia (vide Dr. Stephen Mackenzie in 'Lancet,' 1889) it seems that practice in these cases does not support theory.

*Veratrium Viride* has also been much advocated as a useful drug in cases of eclampsia by three American physicians, Drs. Fearn, Kenyon, and Jewett, and others. The average dose of the fluid extract hypodermically, is ten minims, repeated in half an hour, though larger doses may be given; and Dr. Jewett says, "I prefer to place the patient at once under the influence of the drug fully. The guide to the dosage is the frequency of the pulse; experience seems to justify the statement that no convulsion will occur while the patient is under veratrium sufficiently to hold the pulse below sixty to the minute. The average time required to develope the full effect of a single subcutaneous injection is thirty minutes." "If necessary, smaller doses may be repeated, but I seldom find that the total dosage exceeds twenty to thirty minims of the fluid extract."

I have no experience of this drug; none has been used at Guy's, nor, as far as I am aware, at any other hospital in England, or by English physicians; but I should think, from the above-quoted statement, that it is a drug well worthy of bearing in mind.

Dr. Barnes suggests *nitrate of amyl*, but only on theoretical grounds. *Nitro-glycerine* has been used (in a case reported by Mr. Green in 'B. M. J.,' 1882); and Murrell and Ringer have suggested the use of *nitrate of sodium*, which has properties similar to the above.

Of later years *pilocarpine* given hypodermically has been much used in eclampsia. It was, I think, first brought to the notice of the profession by Braun of Vienna, and a considerable amount of success seems to follow its use. In the last four years I have used it myself in two cases, and have notes of four other cases, and of the six cases thus treated only one died. Of course this is a very small personal acquaintance with the drug, but many other observers speak equally favorably of it.

Last year Fourier, of Paris, recorded a case in which

bleeding and chloroform failed to control the convulsions, but $\frac{1}{3}$ gr. of nitrate of pilocarpine given hypodermically had the desired effect. Five doses were given in thirty-six hours.

The usual dose is from $\frac{1}{6}$ to $\frac{1}{3}$ gr., and it may be repeated at intervals of a few hours, according to the amount of diaphoresis, and benefit obtained. In one of my cases, the patient having previously a hot dry skin, and being so drowsy that it was well-nigh impossible to rouse her at all, sweated profusely after the first injection, and on my seeing her two hours later was quite sensible, and "felt better than she had done for some time."

I may just remark that it is necessary that the solution of this drug should be freshly made, or that the soluble tabloids should be used, as it quickly loses its strength in solution.

Dr. Galabin states that pilocarpine is thought by some to act favorably by diminishing arterial tension; but as it is not nearly so efficient in this way as nitrate of amyl or nitro-glycerine, drugs which have no good effect apparently in eclampsia, he comes to the conclusion that "it must be accepted as an empirical result that pilocarpine appears to tend to prevent the recurrence of the convulsions."

I think that if we accept the uræmic theory of eclampsia the good effect of pilocarpine can be explained differently, viz. by producing a copious diaphoresis and so relieving the kidneys, to say nothing of removing some of the retained excretions directly by the skin.

There are objections to the use of pilocarpine if it be given when the patient is in a condition of coma, because in this case there is loss of consciousness and of reflex irritability. The reflux of abundantly secreted saliva, and the copious secretions of the mucous membranes lining the air-passages, might lead to dangerous symptoms of suffocation. I know of one case of this kind which occurred at the Middlesex Hospital a year or so ago, and Sänger, Spiegelberg, and others speak of similar cases. Fordyce Barker says this drug is too depressing.

*Citrate of caffeine* has been used by one observer, Bolton Corney ('Practitioner,' 1889), with success.

Hot baths to produce diaphoresis and to relieve the kidney congestion may be of use in some cases.

The inhalation of oxygen gas has been tried in two or three cases with doubtful benefit.

*Postural treatment.*—Lastly, Grailey Hewitt, and Routh, believing that disturbances of renal and abdominal circulation from pressure of the gravid uterus, exercised a powerful influence in producing eclampsia, have in several cases placed the patient in the knee-elbow position, maintaining this as long and as frequently as possible; and they say that the patients seem to derive marvellous benefit from this position. Löhlein, too, I believe, has spoken favorably of this treatment.

Dr. Playfair has in one case tried compression of both carotids during a paroxysm, which by producing anæmia of the brain seemed to stop the convulsion, and he suggests that this is a simple method worthy of trial in all cases. Anæmia of the brain stopping convulsions is rather opposed to some authorities, who regard cerebral anæmia as their cause.

*Obstetrical treatment.*—With regard to the question of interfering with gestation, Drs. Robert and Fancourt Barnes say, "The disease depends upon gestation; can the system bear the double strain of gestation and the disease, working, as it must do, with organs which have proved unequal to the task? The question is frequently answered by nature; labour comes on spontaneously."

We would say—

(1) "That in every case in which convulsions have set in premature labour should be induced."

(2) "That when there is marked albuminuria, with œdema, difficulty of breathing, a quickened pulse with a rising temperature, or amaurosis or any form of paralysis, and relief does not follow bleeding, purging, and sedatives, the operation of induction of labour should not be delayed."

(3) "When the patient has had albuminuria in previous gestations, or is known to have Bright's disease and hypertrophied heart, again no time should be lost.

"Conclusive arguments are based upon the following considerations, which apply to all the foregoing three conditions:

(1) "If the case be allowed to go on, even if the woman

recover, every day adds to the strain on the kidneys and eyes, and may lay the foundation of permanent disease."

(2) "It is not justifiable to let the woman run the risk of losing her life or of drifting into grave disease under the expectation of saving the child."

(3) "Granted that the object of getting a live child should rule our judgment, the prospect is better of getting a live child in a future pregnancy than by trusting to the actual pregnancy already gravely threatened."

(4) "The fate of the child is linked with that of the mother, as too commonly, if the mother perishes, the child dies also."

(5) "We are doubly bound to seek our motive of action in the interest of the mother.

"Finally, admitting that some women have, under our own observation and sanction, gone the full period of gestation, and have been delivered of living children apparently without damage; frankly speaking, we would not, with enlarged experience, again encounter a responsibility so great."

Statements like this, coming from such authorities as Robert and Fancourt Barnes, deserve the very fullest consideration. At the same time other observers say that if there are only a few fits, and these are unattended by coma of any length, it may be sufficient to give purgatives, chloral, and bromide, to put the patient on a milk diet, and watch carefully the proportion of albumen in the urine.

The objections to inducing premature labour are that the convulsions may subside and the case go on to the full time of gestation, as did Dr. Herman's case above mentioned, and induction of labour may of itself increase the condition of nervous irritability. Each case must, I think, be treated on its own merits.

# A RETROSPECT IN URETHRAL SURGERY.

By C. H. GOLDING-BIRD.

A RETROSPECT in a branch of surgical practice extending over eighteen years may not prove altogether uninteresting to readers of the 'Guy's Hospital Reports.'

It will point out nothing new; yet it enables definite conclusions from actual experience to be drawn, which may serve as guides to those who are working in the same field.

I have, therefore, carefully gone over the notes of those cases in urethral surgery in which I have had to operate for retention of urine, extravasation of urine, rupture of the urethra, and chronic stricture—in all nearly eighty in number; but I have not included cases of mere catheterism, nor the very few in which this has been supplemented by internal urethrotomy.

The two first conditions mentioned, viz. retention and extravasation, are those for which assistance is mostly sought on the sudden; when the line of practice has to be settled upon and its performance frequently undertaken without much time being lost; it is, therefore, desirable that the surgeon should have clearly before him the alternatives of which he may avail himself, and be able quickly to determine which of them he will elect to adopt.

### *Retention of Urine.*

*Puncture per rectum; supra-pubic puncture; supra-pubic drainage.*—Retention can be relieved in four ways: (i) By catheterism; (ii) by puncture of the bladder; (iii) by puncture of the bladder combined with drainage; (iv) by urethral incision combined with perinæal drainage.

The first of these does not now concern me; yet why it is sometimes so difficult of performance in spite of the skilled hand manipulating perfect modern instruments, is due to a condition of the urethra not sufficiently taken into account, indeed, not generally understood. If we can enter the stricture we can, with patience and time, traverse it with a catheter; but now and then the catheter totally fails to enter the mouth of the stricture, even when, judging from the size of the stream passed and other circumstances, an instrument small enough to be expected to do so has been chosen. Perhaps the commonest cause of this is the presence of one or more false passages which present a readier route; but the one that I refer to I remember many years ago having pointed out to me when studying in Paris. I was being shown through a "Desormaux" endoscope the mouth of a stricture, and the appearance was very marked. The exit or distal mouth of a urethral stricture is situated on a little central papilla of very vascular mucous membrane, produced partly by congestion and partly by the expulsive efforts of the patient, and to hit off the centre of this eminence with the catheter, and so enter the stricture, is as much a matter at times of good luck as ever it is of skill; and hence the suggestion that a large open-ended catheter should be passed down first to fill up the surrounding valley, and then a fine catheter should be passed down the calibre of this open-ended one. Though I have several times tried this plan, I never found it any material aid.

Leaving, then, catheterism, I will speak of the operative relief of cases of *uncomplicated retention;* that is, where there is no other condition co-existent, such as extravasation, which would by some necessary operation for itself relieve the retention at the same time Curiously enough,

where extravasation exists we find retention rarely a prominent symptom, the extravasation having apparently taken off the stress from the bladder walls.

Everyone is familiar with the fact that in old prostatic cases great distension may take place without the patient being aware of it, provided he can only take off the "last straw" of bladder stress by passing his usual tablespoonful at frequent intervals; and twice in little children, four and five years of age, have I seen the bladder kept enormously distended without any distress, and never thoroughly evacuated, from the dread of the pain that they knew would ensue, if the bladder were emptied upon a vesical calculus, which in each case was present; and in one, even after removal of the stone, it was a long time before the child could be educated once more to micturate properly at usual intervals.

Years ago Mr. Cock introduced his once popular *puncture per rectum;* and in the seventies I remember how constantly it was employed here, when, by-the-bye, retention cases from failure to pass a catheter were very much more frequent that nowadays. The last case in which I employed it was in 1881.

The instrument and the method of using it were familiar to past Guy's men; but now many may never have even seen it. In my opinion it has had its day, and as I am going to give my reasons for this opinion, I must first mention its main characteristics. The instrument is a long, curved, silver cannula ending in an oblong shield, and carrying either a blunt or a sharp trocar. The patient being in the lithotomy position, the cannula, having the blunt trocar inserted, is passed into the rectum, guided by the left forefinger, and made to impinge against the anterior rectal wall immediately behind the prostate. Held firmly thus by the left hand the blunt trocar is withdrawn and the sharp one is introduced, and then the instrument is thrust upwards and forwards through the rectal wall into the bladder. The cannula is eventually tied in, the shield resting against the anus, and a tube being attached, the urine can drain away *per anum* into a suitable vessel.

My experience of this operation is against its continuance,

in the presence of an easier and safer method of puncture; and putting aside the fact that it was by no means so easy an operation as might from description appear, it was one that at times failed, and was not free from risk.

The risk lay in the fact that where the prostate is enlarged, it is frequently impossible to feel its posterior limit, and so the puncture having to be made at a point where the prostate was believed to end, but beyond the reach of the guiding forefinger of the surgeon, there was danger of the peritoneum being wounded; and it is a surprise to me that this accident did not more often occur. Once I saw this done with a result that can be well imagined. Twice I have known puncture *per rectum* quite fail; in one case blood-clots lay on the floor of the bladder, forming an insurmountable barrier to the flow of urine through the cannula. In another the failure was discovered at the autopsy to be due to a mass of malignant disease, through which the instrument was not long enough to perforate.

My experience of *supra-pubic puncture* for retention is very different. I have never seen any accident with it, immediate or remote, in my practice; it is as simple an operation as it is perfect, and with the most ordinary care entirely devoid of risk. We must, in estimating its usefulness, distinguish between supra-pubic puncture and supra-pubic drainage; in both the puncture is the same, but the after-treatment different, and each has its appropriate value.

*Supra-pubic puncture* for retention may be done either with an aspirator, or with a trocar and cannula. For many reasons I prefer the latter. The expulsive efforts of the bladder will always drive out as much urine as an aspirator can safely draw; whilst the size of the aspirator needle is generally so small, that should the urine prove phosphatic and ropy great difficulty in the evacuation would occur. This once happened to me in the case of a man only aged thirty, and though a large trocar (not the aspirator) was used (No. 6, cath. gauge), the stinking, ropy, phosphatic urine would hardly flow after the first expulsive gush was over; with an aspirator needle I should probably have failed in relieving the patient.

The kind of case in which I have used simple puncture

of the bladder, the cannula being withdrawn after the evacuation, is that where the stoppage is sudden, recent, and temporary, perhaps due to cold or alcohol, in a man who is habitually able to relieve himself, though the subject of chronic stricture or enlarged prostate, and in whom attempts at catheterism are extremely painful and accompanied by bleeding from the very turgescent state of the mucous membrane.

If the case is one of stricture, I regard supra-pubic puncture as fraught with less risk, suffering, and certainly less delay, than the giving of an anæsthetic, with perhaps half an hour's patient attempt to get a small catheter through the stricture. The bladder having been emptied by puncture, and a dose of opium given, after a good rest the man will certainly relieve himself naturally, as he has been accustomed to do; the treatment of the cause of the stoppage there is then time to consider.

I have punctured also in cases of enlarged prostate, where from the same causes mentioned just now temporary stoppage exists; but these are not common. Simply enlarged prostate is no bar to catheterism; but I have employed puncture where, from one cause or another, urethral hæmorrhage and perhaps false passages have been the accompaniments. Once, however, I punctured the bladder in a case of recent gonorrhœa with acute prostatitis in a young man, but it was in 1879, and puncture *per rectum*, not supra-pubic, was performed.

In such cases as this last, which are unusual, catheterism is from the state of the parts the least desirable method of relief. And in all the cases to which I have referred, and which form a class by themselves in respect of acute retention and temporary inability to be relieved by the catheter, I think supra-pubic puncture most valuable, practically free from risk, and in the future it leaves the patient with no complication, such as might arise, and does occasionally, where ill-judged and repeated attempts at catheterism injure the urethral canal.

*Supra-pubic drainage* is an expansion of the mere puncture, but the puncture must be done with a fairly large cannula, and immediately the trocar is withdrawn, a Jacque's flexible

catheter (I prefer No. 9) must be crowded into the bladder up to the hilt; the cannula is now withdrawn, and subsequently so much of the catheter that only two inches remain in the bladder. Through this the urine will flow away, and a drainage-tube being attached the urine may be continuously siphoned off as long as is necessary. I need hardly say that to put a peg in the catheter might lead to an extravasation of urine around the puncture.

This operation is a very favourite one with me, and is suitable where not only has the retention to be at once relieved, but absolute rest given to the urethra before again attempting catheterism; or where for any reason it may be well to divert the stream from its natural course for a time. I have employed it, therefore, in old or complicated strictures, where from the urgency of the distention, together with the presence of urethral hæmorrhage, laceration, or false passages, catheterisation has not been possible or feasible; and where from the character of the stricture it is a matter of clinical knowledge that mere puncture will not suffice for permanent relief. I have also employed it to give relief to immediate distention and to divert the water-course in two cases of ruptured urethra, in one of which the puncture stood me in good stead in subsequent treatment, as I will narrate presently.

This operation is on all fours with tracheotomy for a stenosed larynx, or colotomy for a strictured rectum, where not only is relief at once afforded, but the rest given to the natural exit in both cases has a valuable, though in some cases only temporary, therapeutic action.

After the operation a catheter in the most obstinate cases can be passed *per vias naturales* in three to seven days; only once did I have to retain the drain up to the twenty-first day. The puncture invariably heals at once and soundly.

There is yet another way of puncturing the bladder, or rather the membranous urethra—by *Cock's operation*—a form of perinæal section, but this will be referred to in another connection. I must here, however, express my firm conviction that it is not well to open the urethra for mere retention. Once introduce scar-tissue into the urethral wall, and subsequent contraction may occur; and if the urethra, save for

the submucous thickening that forms the stricture, is sound, let us keep it so by all means. Still, if the urethra must be opened, there is less to be said against incising the membranous than any other part of the canal.

### *Extravasation of Urine.*

For clinical purposes we must distinguish two classes of cases under the term *extravasation* of urine, viz. (1) those depending upon stricture or ulceration; (2) those depending upon rupture from external violence.

To take the first of these, what may be called "ordinary extravasation." It is in these cases that perinæal or urethral, rather than vesical, surgery is required, the choice of operation being guided by concomitant circumstances. A clinical study of 27 of these cases of extravasation on which I have operated reveals: 17 due to stricture, 3 due to impacted stone, 3 due to phimosis, 1 to congenital narrowing of the meatus urinarius, 2 to recent gonorrhœal ulceration, 1 to extensive and extending syphilitic ulceration of the rectum involving the urethra. Clearly, with so many different causes, the method of treatment must vary.

In *stricture-extravasation* the surgeon has three objects to effect: 1. Removal of infiltrating urine from the tissues; 2. The immediate and continued evacuation of the bladder; 3. The cure of the stricture.

To accomplish all these at one operation is hardly possible, and in my opinion rarely advisable to attempt, and that not from any operative or mechanical impossibility, but because, as a rule, the urethra is in such a state from septic inflammation, that the best results in the end are obtained by dealing with the stricture separately; for the moment it is indeed of secondary importance. For these reasons those perinæal operations whose main object is the treatment of the stricture by division on a guide, as Syme's or Wheelhouse's operation, I do not adopt in extravasation cases, in which I have done a Syme but twice in seventeen times; and though in both I divided the stricture by external urethrotomy, I did not keep the catheter in the divided stricture, but passed it through the perinæal wound into the

bladder, thus obtaining good perinæal drainage, of which I shall speak directly. In both cases the extravasation was limited, chronic, and in the form of urinous abscess. Besides, it is the exception to be able to pass an instrument in extravasation, and hence some method of relief must be adopted which does not depend upon a sound being first passed as a guide ; and hence it is why merely incising the infiltrating tissues is often resorted to.

Once only (July, 1876) did I rely upon merely incising the extravasated tissues, trusting, as a high authority has put it, that urine that can find its way into the tissues can find its way out through incisions into them. So it does, but by thus infiltrating the tissue recovery is retarded, the danger of septic absorption great, and the bladder is not completely evacuated, sufficient obstruction being met in the tissues to retain always a fair amount of urine in the bladder. The after-dressing in mere incision is most difficult, and a sore sacrum from the trickling back of urine it is hard to avoid, whilst there is danger that urinary sinuses will be left in the perinæum or scrotum. In a word, though a recognised method, it is not indicative of a high order of surgery.

The secret of success in extravasation cases is the immediate draining of the affected tissues, and this free incisions will do, and then by efficient drainage of the bladder to absolutely prevent any fresh extravasation.

To incise the scrotum and perinæum and then puncture above the pubes will no doubt appear an easy and efficient method, but I have a reason why I have never adopted it. I object to making two incisions where one will do, and, what is more to the point, one does not always find in extravasation a bladder so distended as to make the suprapubic puncture as free from risk as it is in cases of simple retention.

Further, where it is even possible to pass a fine sound as a guide, I yet do not prefer to perform either Syme's or Wheelhouse's operation for the mere drainage of the bladder in extravasation of urine, and for this reason, that it is the rule and not the exception for a stricture, when once the water-stream has been diverted, to yield sufficiently to allow

the passage of a catheter and subsequent treatment by dilatation; hence the incising of the stricture is doing more than most cases require, whilst it introduces a wound into the urethra, which will heal by scar tissue, and may by its contraction add considerably to future difficulty in curing the case. I believe it to be the wiser and sounder practice to cut down on the sound (supposing the case where one has been passed through the stricture) in the neighbourhood of the prostate, that is, in the membranous urethra, and thus, as in Cock's perinæal operation, to drain directly from the prostatic urethra, leaving the spongy urethra and its stricture alone. Anatomical considerations, apart from clinical observation, would suggest this course. Stricture of the urethra behind the bulb is unknown, and certainly I have never myself met with it as the result of Cock's operation, in which the membranous urethra and the tip of the prostate are incised, even where a catheter has been worn for weeks in the perinæum; yet all the conditions are present to favour the formation of a cicatricial stricture. The explanation lies in the simple fact that there is no spongy tissue around the membranous urethra; and a scar in mucous membrane is anywhere of little or no moment, but it becomes a matter of importance if the "submucosa" is involved; for in this the cicatricial tissue is formed: and where there is no submucosa to speak of, as in the membranous part of the urethra, a wound of the mucous membrane is of no account.

Believing, then, that to operate behind the stricture is the best method in cases of extravasation, and that operations on the stricture for this condition do more than is required at the time, there remains but one form of operation that fulfils all requirements, and that is to open the membranous urethra as near the prostate as possible. It is this operation that is known to all Guy's men as Cock's operation, and was first described by the late Mr. Cock in these Reports for 1866. It is an operation little known, or at least little appreciated by surgeons as a rule, and though I have not unfrequently heard speak of Cock's operation having been done, I have as frequently found that any cut in the perinæum, where a catheter cannot be passed, is called

by that name; whether the urethra—and so the bladder—is reached or not.

There was an older operation on somewhat the same lines—at least as far as the early steps of the operation were concerned, and it may have given the idea of the operation I am speaking of. The older operation was also for impassable strictures—impassable to catheters of the day—and the surgeon was directed first to pass an 8 silver catheter to the stricture, then to stab with a knife the urethra in front of the prostate, and having done this, to prolong the incision anteriorly and upwards, dividing the brawny stricture completely throughout its entire length. The silver catheter was then passed on through the incised tissues into the bladder. The full details of Mr. Cock's operation will be found in the volume just referred to; they may be thus summarised. The patient being in the lithotomy position, the left forefinger feels in the rectum for the tip of the prostate, or if there is much pre-rectal extravasation the middle line must be judged of rather than felt. A broad double-edged scalpel is now thrust into the perinæal raphé half an inch in front of the anus until it reaches the tip of the prostate as determined by the left forefinger. The blade is inclined a little laterally at the last part of the thrust to to make the opening of the membranous urethra more certain, and this is further assisted by the fact that the urethra at this point is dilated, being behind the stricture. On removal of the knife a broad-grooved director is passed along the track into the bladder, and along this again a 12 English catheter, previously straightened out. A tube is now fastened to this catheter as it protrudes from the perinæum—it being tied there firmly—and so the urine runs off as excreted into a suitable receptacle. Scrotal incisions are made as usual. Now such an operation presents advantages which are offered by no other that I know of. All the conditions necessary for uncomplicated recovery are fulfilled, and that with the least possible amount of interference on the surgeon's part. The urine can no longer escape into the tissues, the bladder is completely emptied and maintained so; drainage is from the lowest point—and in that respect perinæal drainage is always better than either penile or pubic drainage; the

spongy urethra is not interfered with, only the membranous, in which, as above explained, there is no fear of cicatricial contraction resulting, and the stricture is insured absolute rest and placed under the best conditions for eventual treatment by dilatation, which in the case of the spongy urethra is a better method of treatment than any form of urethrotomy; and lastly, the surgeon needs no guide other than the left forefinger in the rectum in the performance of the operation.

By the fifth day the perinæal track will have been well established so that there is no difficulty in changing the catheter if necessary. The length of time that it is worn will depend upon the time that it takes to re-establish the penile route, and for the infiltrated tissues to recover; but where retained for weeks no disability results, the perinæum heals soundly, and the membranous urethra does not suffer from the prolonged presence of the catheter.

The stricture, if one be present, can, whilst the extravasated tissues are recovering, be dilated by the passage of bougies down as far as the perinæum; and when the dilatation has reached the size of a No. 7 catheter (English gauge), it is easy to pass a catheter *per penem* all the way round (the perinæal drain being removed) and tie it *in situ*.

Perhaps the reason that Cock's operation is not more general is the uncertainty about its performance. It is easier certainly to describe than to do, and there must be just an element of uncertainty in such cases where there is much pre-rectal extravasation obscuring the position of the apex of the prostate. Yet here the difficulty is more apparent than real, since it is in these very extreme cases that distension of the membranous urethra by retained urine may be expected, and so the chances of the knife puncturing it are increased rather than diminished. Where the perinæal structures are sloughing, even gangrenous, though the urethra can be opened by the sharp knife, there may be difficulty in introducing the blunt director in its track, and though it has never occurred to me not to open the urethra, in one case I did fail to pass a catheter into the bladder. The case is of some interest and will bear repeating. It was that of a tradesman in South London, who at twenty-four

years of age had been under Mr. Cock's care for stricture after gonorrhœa, and Mr. Cock informed me he had never succeeded in getting through the stricture, which fact alone I think makes the case all but unique. I saw him in 1879, at thirty-six years of age, with extravasation and gangrene of the perinæum and in an exceedingly marked typhoid state. I learnt that all these years the urine had been passed only in a very fine stream, and that gradually lately swelling had commenced in the perinæum and advanced to the present state of things. To pass a catheter was impossible, and I therefore performed Cock's operation, and urine flowed through the incision; several attempts to get the catheter through the wound into the bladder failed, the tissue being in a sodden gangrenous state. So hot fomentations were applied, and the urine was allowed to drain freely into the dressings. In this condition he lay some time, his general and local state rapidly improving, and in five weeks I succeeded in passing a No. 1 catheter through the stricture. Dilatation was gradually carried on, and when a No. 7 was reached it was passed into the bladder and drainage established *per penem*. After this he made a complete recovery, and remains well to the present time, but once a week he passes a full-sized catheter, retaining it a short time—at least he was doing so two years ago.

Beside the point in connection with the operation, the case is a good example of the marvellous way in which a stricture will yield when the water-stream is diverted, for we have undoubted testimony both of the chronicity as well as of the severity of the stricture. I would further quote this as corroborating the opinion I have already expressed, that in extravasation it is best to leave the stricture alone, and to confine operative measures to behind the bulb.

Out of fourteen cases of stricture-extravasation in which I have performed Cock's operation, I have seen but one death attributable to the operation. It was in the case of a man aged thirty-seven, who at the time of the operation bled profusely from apparently a wounded vein. I have never known of a similar mishap; it must be looked upon in the light of an accident rather than a probable complication of, and therefore argument against the operation.

In four other cases that ended fatally the deaths were due to the usual urinary causes, viz. surgical kidney and uræmia, but in no way to the operation; the patient in each case was already seriously ill when first treated.

It is in cases of acute and urgent extravasation that the brilliancy and high value of this operation appears. The patient being placed within a few minutes in a position of ease and safety, with a minimum amount of disturbance of either himself or his tissues.

I may here draw attention to the irregular way, if I may so call it, in which the tissues behave when acutely infiltrated with urine. Often the suffering is severe, and the scrotum rapidly becomes hot, red, and eczematous, or even gangrenous; but the reverse of this is not uncommon where the extravasation causes no pain, no febrile disturbance, and scarcely, if any, local evidence of inflammation, so that the case seems one of simple passive œdema. Thus in my notes I find, for instance, one case in which an old man of seventy-one walked about quite unconcernedly with extreme extravasation for a week, only applying for relief because at last he could not micturate; his tissues being as yet uninflamed, and presenting the appearance of simple, though extreme, œdema; and another in which a man of forty-five ruptured his urethra, and for seven days was about with extensive urinous œdema, without pain, and without any inflammation.

This inactivity of the extravasation often leads to doubt as to whether it is not merely œdema from some general cause, or from an attack of erysipelas; and when we remind ourselves that urinary extravasation may occur without urethral stricture, the diagnosis becomes even more puzzling.

As to the *causes of extravasation*, in by far the majority of cases there exists of course some mechanical obstruction to the outflow of urine, which brings about ultimately its extravasation; indeed, so seldom is it otherwise, that the exceptional cases may fairly be taken as proofs of this general law. Examples of cases in which mechanical obstruction more or less existed, yet not ordinary urethral stricture, and on which I have had to operate on account of extravasation, are as follows:—Three cases due to phimosis at the ages of fifty, forty-two, and eleven and a half years. The phimosis

in the first case was from commencing epithelioma, in the second from adherent and contracted prepuce after repeated balanitis, and in the last case the phymosis was congenital. Once also have I seen it from congenitally narrow meatus urinarius in a man of thirty.

In this case the extravasation, though preceded by a pain referred to the perinæum, was seen—since it occurred whilst he was under observation—to commence at the level of the corona, and to extend backwards till the whole scrotum was involved. At the operation, after division of the meatus, a No. 12 catheter was easily introduced down the penis. There was nothing wrong with the perinæum, though the patient had felt the water-stress mostly there, and the extravasation clearly started immediately behind the glans penis.

Then extravasation from impacted calculus is occasionally met with in children. Twice, at the ages of twenty-five and twenty, I have seen extravasation result from and during an attack of recent gonorrhœa. Where no stricture whatever existed in one of the two, the history was that, two weeks before, whilst injecting, a sharp pain was felt in the perinæum, and the extravasation shortly after was noticed. In each a large urinous abscess occupied the perinæum, which, when opened, showed the bulb "dissected out" by the abscess; a No. 7 catheter could in each instance be passed, and in each was tied in, for the most careful examination of the exposed urethra failed to detect any opening in the urethra, and therefore it was not incised. These two cases were evidently due to gonorrhœal ulceration of the floor of the urethra, coccus-migration into the tissues, together with slow extravasation of urine, accelerated or even caused in one case by syringing.

I have notes also of two cases—aged thirty and thirty-five—where in one the extravasation came on three weeks after a false passage had been formed, yet there was no obstruction, for when the man came under my notice I passed a No. 7 easily. In the other case the patient was wearing a No. 4 catheter for dilatation of a stricture at the time the extravasation began to appear. In these also the extravasation was not due to undue force to overcome an obstruction, but to a pre-formed breach in the mucous membrane.

These cases of extravasation in which no bar exists to the passage, at the time of the operation, of a large catheter can be with safety treated on somewhat different lines. The perinæal drainage offered by Cock's operation, theoretically and practically the best for relieving the bladder and preventing further extravasation, is not absolutely necessary under the condition above-mentioned; for where a No. 8 catheter—or higher number—can be passed *per penem*, it having such a large bore offers no noteworthy obstruction to the flow of urine, provided that it is never plugged, but attached to a syphon drainage-tube whereby the bladder is constantly drained. Hence simply incising the extravasated tissues is the only operative measure necessary, and the recovery is correspondingly accelerated; yet in all these cases to perform Cock's operation to insure perinæal drainage would be perfectly justifiable, and is the constant practice of some surgeons, yet in my opinion it is not as a rule necessary.

If there were reason for believing that the urethra was severely ulcerated, and that the presence of a catheter would be injurious, then perinæal drainage would be called for; but the merits of each case must decide what step is taken; still, having several times successfully adopted penile drainage over perinæal, through a large catheter, merely incising the scrotum for the extravasation, I have come to regard a perinæal section as *de trop*, at least unless penile drainage has been tried and found insufficient.

*Ruptured urethra; rupture-extravasation.*—Whilst for the most part cases of rupture-extravasation might be classed under the same head as the stricture- (or obstruction) extravasation cases, yet many of them present special features, which are sufficiently important to allow of their being separately considered. The rupture is not, like stricture, confined to the spongy urethra, though where due to direct violence and not to fractured pelvis, the injury is likely to be anterior to the triangular ligament; it may be complete or incomplete, and where the former, the separation of the two parts of the spongy urethra is likely to be considerable, even three quarters of an inch; it may be complicated by fractured pelvis whether caused by it or not, or by internal injury,

rendering the case a more serious one than it otherwise would have been. These considerations are necessary to show that the nature and extent of the original violence must weigh with the surgeon in determining the steps he will take in regard to his treatment of the urethra, and of the extravasation, if present.

Extravasation is not a necessary accompaniment of ruptured urethra; the presence of this complication being in part determined by the amount of urine in the bladder at the time of the accident, and partly by the length of time that elapses before the patient is seen or the nature of the accident recognised; further, the presence of extravasation may be masked for the time by great blood effusion, especially where direct violence has caused the rupture. Great tearing of the perinæum seems at times to close the urethra by "torsion," so though the bladder is distended no urine may escape.

A classification of ruptured-urethra cases might be made from their mode of origin or the subsequent local conditions; but my present remarks being made from the purely clinical aspect, it will be better if I classify them from point of view of treatment.

From amongst my notes I instance the following cases; for some clinical details must be given to justify the operative steps that were undertaken, and which differed in each case.

Case I.—A case (September, 1881) in which attempts at catheterism failed, and I had to cut down on the rupture, guided by a staff passed as far as it would go. It was a case of ruptured urethra produced a few hours before by a railway-smash, the pelvis being crushed between an engine and a wall. My note at the time was to the effect that the perinæum was not bruised, the bladder was distended, but that there was no extravasation, the pelvis was broken, and from the collapsed state of the patient further internal injury was suspected. On opening the perinæum I found the bulbous urethra torn across, the ends being separated by an interval of an inch. I passed *per penem* a No. 11 catheter, guided it through the proximal end of the urethra, and so into the bladder. The ends of the urethra were then

stitched over the catheter, which was retained *in situ*. The patient, however, never rallied from his collapsed state.

The urethra in this case had been most probably cut through by the scissor-like action of the two ascending rami of the ischium, for the severity of the accident and extent of injury to the bones rendered it likely that the laceration had been thus caused. Whether or not this was so, there was a clean tear through the urethra without the extreme bruising of the perinæum seen in direct external violence, and hence the case was most suitable for adaptation of the urethra over a full-sized catheter. It is a question whether a stitch is even necessary, and there are theoretical objections to its employment, yet I know of no harm having resulted from the use of a suture either in my own practice or that of others; whilst the great advantage of it is that it holds the ends together in the early days of repair, so that if the catheter has to be soon changed there is less chance of its missing the proximal urethra, and of making a false passage.

Two cases in which I passed a catheter all round at the first attempt, thus suggesting that the rupture was partial.

In Case II (September, 1887, aged forty-five) there had been direct perinæal violence a week before, and he really applied for relief because of increasing difficulty in micturition which ended in complete retention. During the week the patient had observed increasing swelling in the perinæum which spread to the external genitals, and when seen these parts were greatly swollen with clear, non-inflammatory, urinous œdema. He made light of his condition, had no pain, and seemed quite well in himself. I passed a No. 11 catheter the usual way, incised the swollen parts, and he rapidly recovered. The cause of the retention was not due to excessive damage to the urethra but, I believe, to the gradually increasing extravasation, which mechanically closed the urethra by pressure.

Case III (September, 1886) was very similar; in that there was extravasation and retention, but the perinæum was very ecchymosed. The patient was a child of nine years old, and three hours before had fallen astride a bar.

I passed a No. 6 catheter all round, incising the extravasated tissue; all went well afterwards.

In such comparatively simple cases, where a catheter can be at once passed, it is fair to assume that only partial rupture of the urethra has occurred, and provided the catheter can be retained without changing until tolerable union has occurred, say seven to ten days, there is little likelihood of any subsequent difficulty arising. Indeed, in all cases where a catheter is passed all the way round, whether the perinæum is opened or not, this rule still holds good. In such cases also as these two it would have been, in my opinion, unwise to perform Cock's operation with the view of draining the bladder the more readily; for it is my experience that provided a full-sized catheter can be worn, siphon-drainage by its means is quite effectual.

In three other cases I performed Cock's operation, preferring to open the sound urethra behind the rupture, on account of the unhealthy or damaged state of the injured portion.

Case I was that of a workhouse inmate, aged fifty-five (March, 1881), who was seen four days after a blow which caused extensive bruising in the perinæum, and extravasation. There was, when he was admitted into hospital, a prominent and hard urinous hæmatoma occupying the perinæum, and the tissues were in such a state that even had the urethra been found, I believed it to be in an unfit condition to allow of a catheter being retained in it—still more unfit to allow of any stitching together of torn ends. I, therefore, at once did a "Cock," and blood and urine came out through the incision, but I failed to get a catheter into the bladder. The surrounding tissues were incised. On the second day an anæsthetic had to be administered for the further opening of an abscess that had formed, and the patient died on the table presumably from the chloroform. A post-mortem inspection showed the bulb completely torn across; but the urethra had been opened at the operation in the usual situation, *i. e.* the tip of the prostate. The bar to the passage of the catheter I attributed to the very bruised and sodden state of the perinæal tissues.

Case II (May, 1884) was that of a man, aged 54, who fell astride a bar six hours before he came under observation.

For half an hour he had pursued his business, and then walked up unaided to the hospital on account of extreme distress from retention and inability to pass any urine at all. Bruising was severe in the perinæum, though the pain was quite moderate, but there was besides extensive extravasation of urine. The bladder was fully distended to the umbilicus. A catheter failed to enter the proximal end of the urethra, and, taking the lacerated state of the tissues into consideration, I at once performed Cock's operation, putting in a No. 12 as perinæal drain into the bladder. No attempt was made to search for the point of rupture, but the necessary incisions to relieve the extravasation were made. In three weeks a No. 11 catheter was passed all the way round, and six months later the patient reported himself as perfectly well; but he passed a catheter once a week as a precautionary measure.

The successful end of this case may be taken as justifying the means employed; still it is quite an open question whether the ends should not have been searched for, and an attempt made to unite them at once. The early date on which it was possible to pass a catheter *per penem* suggests that the opening made by the knife could not have been far from the proximal end of the urethra; had it not been so, greater difficulty in establishing the natural passage might have been expected.

Case III, aged 41 (June, 1883), was almost identical in history with the last; six hours had elapsed before his admission, he walked to the hospital, his chief complaint being of retention which was complete. The state of the parts was as in the last case.

In this case I determined, whilst getting a perinæal drain, to utilize the proximal end of the urethra instead of making a fresh wound, and so guided by a staff *per penem* I opened the perinæum, found the proximal urethra, passed a large catheter through it into the bladder, retaining it there *in situ* for a week. I then passed a catheter all the way round, and a fortnight later the man was discharged well, and able to pass a catheter for himself.

Quite strictly this was not a Cock's operation, since a fresh opening was not made in the urethra, yet the lines of

treatment were those of Mr. Cock, and hence I classify it as such.

Contrasting it, however, with Case II, it opens up the important and interesting question of whether, when a catheter cannot be passed, and when for any reason the ends of the urethra are not sutured in position, it is not better, in establishing a perinæal drain, to utilise the proximal urethra rather than open the urethra behind it. The mere opening of the membranous urethra does no harm, as I have shown already ; but it is quite conceivable that in cases of rupture there might be a marked interval between the proximal opening in the urethra and the point where the knife entered, and so when the time came for passing a catheter through the penis its route would probably be *via* the distal end of the rupture and then through the operation-opening, thus missing the part of the urethra that lay between this latter and the rupture. Such a state of things does of course now and then happen in severe chronic stricture, where dilatation cannot be carried out and the catheter is tied in, having been passed by the aid of an external urethrotomy the whole way into the bladder. At first the catheter for a short distance lies naked in the wound, and gradually gets hidden as granulation proceeds ; and thus a short passage in cicatricial tissue really lies in the site of the old stricture. Such cases do perfectly well, but require constant after-attention to avoid contraction.

Now where a Cock's operation is done for ruptured urethra this state of things is not unlikely to ensue, and in the case I have quoted I can, as I have just said, only attribute the early cure to the fact that my incision and the proximal end of the rupture were to all intents and purposes identical.

These considerations have caused me to conclude that where perinæal drainage in rupture has to be adopted, it is best done, if the state of the parts and the position of the rupture allow, through the proximal ruptured end rather than through an opening made behind it.

I have notes of two cases in which I did supra-pubic puncture for ruptured urethra. Such a step evidently is only possible when the bladder is sufficiently distended, but

I do not by this at all mean to imply that where this is the case supra-pubic puncture is necessarily the best or only operation. Both cases present peculiarities of great interest.

Case I.—A man, aged 40 (May, 1886), six weeks before admission had fallen astride a bar; he could pass his water afterwards, but it was mixed with blood; he did not think much of his accident, and until he found increasing difficulty in micturition six weeks later did not seek relief. His own medical man tried to pass a catheter, but could not reach the bladder. I saw the patient shortly after with extreme distension, and so performed supra-pubic puncture, with permanent drainage. A few days later, failing to pass a catheter, I opened the perinæum, and found the two ends of the urethra widely separated from each other, and only connected together by a cicatricial cord. A No. 10 catheter was then readily passed all the way round through the penis, and tied *in situ*.

In three weeks' time the patient could pass a stream about half the normal size, and dilatation was commenced, so that when discharged, three weeks later, he was passing a full-sized instrument for himself.

The chronicity of this case, the absence of extravasation or any local inflammation, and the retention being the pressing condition, determined me in puncturing and draining, in the hope that as the patient had for six weeks been able to pass his water, a catheter might be passed after an interval. As, however, that was not possible, the exploratory operation was undertaken, and it is certainly very astonishing that with the separation of the ends discovered, the patient should so long have been able to relieve himself, and not have suffered from extravasation.

Case II.—A man, aged 43 (September, 1882), was crushed by a weight falling on his hips, and when he was admitted there were signs of fractured pelvis, the symphysis was separated, and the perinæum much bruised. A catheter was apparently passed, but no urine was drawn off, nor blood. Twenty-five hours later, being in great distress from retention, I explored the perinæum but quite failed to find in the pulped tissue the proximal urethra, and so performed supra-pubic puncture with drainage. After eight days the supra-

pubic drain was removed, and through the resulting opening an ordinary catheter was passed into the bladder, directed towards the neck of the bladder, and being passed on made its appearance through the unhealed perinæal wound by a reversed process of catheterism. A director was now passed along the track thus formed from the perinæum, and a catheter easily introduced by its help all the way round through the penis into the bladder.

Beyond the general note that "all went well afterwards," I have been unable to trace the case further. Supra-pubic puncture here performed a double part, for being done for retention when the proximal urethra could not be found, it subsequently became the means of finding the occluded passage. That the urethra must have been pretty well "torsed" by the accident is shown, not so much by my inability to find it, as by the fact that extreme distension of the bladder had failed to force the urine through it.

Had a Cock's operation been performed in this case, there can be little doubt that the proximal ruptured end would never again have been "worked into" the general continuity of the newly-created water-way; and that nothing short of what actually was done would have availed again to render it patent.

A curious and puzzling complication occurs now and then in rupture-extravasation, which is determined by the amount of laceration at the time of the accident, whether by direct violence or consequent on fracture of the pelvis. I give an example of each.

Case I.—A child, aged 7 (June, 1883), was crushed, and admitted three hours after with comminuted fracture of the pelvis; great bruising and swelling of the perinæum, complete retention of urine, and bleeding from the penis. Failing to pass a catheter under an anæsthetic, I opened the perinæum, but could not discover the proximal urethra, and so with the finger in the rectum I performed Cock's operation, passing a No. 7 catheter in the usual way to form the perinæal drain, and through it the urine and blood came, and when pushed home it was noticed that it could be felt by the hand placed above the pubes. The amount of urine that flowed away was not as much as expected, but two hours later much urine came

by the side of the catheter, it being presumably blocked by blood-clot. For four weeks all went well; the catheter was occasionally changed, and urine flowed through it, and also by the side of it, but it was now noticed by the dresser that on using the lotion to wash out with, some was returned by the rectum. It had been noticed at the operation that only the rectal mucous membrane remained anteriorly, everything external being torn through at the accident; and as urine had been coming by the side of the catheter, an endeavour was made to establish the penile route, chloroform being given and the perinæum reopened. This not being successful I determined to leave the child passing his water by the new-established perinæal track, without any catheter at all. After two months when consolidation was sound I once more thoroughly examined, following up the perinæal fistula; I then found that it led behind the pubes, where was a cavity bounded in part by the back of the symphysis (which was separated by half an inch, though bridged across with fibrous tissue), and into this cavity the urine clearly drained from the bladder, which could be now made out lying behind, and entirely torn across at the neck from all connections. I now forced in water hoping to distend the bladder, and a dull area was created above the pubes. A catheter now introduced by the perinæum entered this and was tied *in situ*. My next note dates a month later, when it states that the child is now passing water normally by the penis, and he was temporarily discharged from hospital. After a week he was readmitted, and a catheter passed *per penem;* he was kept in for catheterism for three weeks, and then discharged micturating apparently in quite the natural manner.

As the result showed, this was a case rather of bladder torn away from its urethra than of ruptured urethra; and what at the first operation had been taken for bladder was clearly a "subsidiary bladder," a space behind the pubes into which the true bladder drained. As in a case already mentioned, it is to be noticed that complete rupture of the urethra is consistent with retention without any extravasation.

Case II, also illustrating the "subsidiary bladder," is that of a man, aged twenty-six (May, 1881), who was

kicked two weeks before by a horse in the perinæum; blood came from the urethra. After some days he could not micturate, so his doctor passed a catheter and drew off urine. For the next three days he passed urine apparently naturally, then he came to the hospital with retention. The dresser passed a catheter, drawing off over a pint of urine; but he remarked to me later that "he did not think he got into the bladder." The following day I also passed a catheter, but being certain I had not reached the bladder I opened the perinæum and found a large cavity in front of the rectum, surrounded by firm, inflammatory infiltration, in which the urine collected from the proximal urethra which was found ending in it, the rupture having been complete. I succeeded in passing a catheter through the penis into the bladder, and in two months he was discharged, able to pass a soft catheter for himself, the perinæal wound being very nearly healed.

Somewhat similar local conditions may be seen at times in ordinary stricture-extravasation where a large urinous abscess occupies the perinæum; but the clinical history in such cases differs widely from that of rupture cases; and the contents of the cavity is pus and urine in the one case, and in the latter ordinary urine. Indeed, in neither of these two cases did abscess form at all.

### *Chronic Stricture.*

Operations for stricture pure and simple, uninfluenced by any of the complications before mentioned, are, in comparison with those which these complications call for, few, and are still getting fewer. Patients by early treatment are saved from many of the ulterior consequences of stricture, and hence catheterism is rendering urethrotomies uncalled for. So in the course of eighteen years, out of a very large number of strictures that one has been called upon to treat, only just over a dozen have required operation. Under operations for stricture I do not include catheterism or internal urethrotomy; and the only complication which I would classify here with stricture is that of urinary fistulæ, since their treatment rests upon the treatment of the

stricture, and this latter being cured, the fistulæ will generally heal.

The operations open to surgeons, and which my cases illustrate, are those of Syme, Cock, and Wheelhouse, and the old "Boutonnière" already referred to; and the choice of one of these in any case will depend upon the passability or not of the stricture; and whether it is thought well or not to operate on the stricture directly.

Hence two classifications are possible:

1. Classification from point of view of the disease (stricture).

| | |
|---|---|
| Operations on the stricture itself. | 1. La Boutonnière. |
| | 2. Syme. |
| | 3. Wheelhouse. |
| Operations on the unstrictured urethra near prostate to give rest to stricture. | 4. Cock. |

2. Classification from point of view of the operation.

| | |
|---|---|
| Operation with guide *to* stricture. | 1. La Boutonnière. |
| | 2. Syme. |
| Operation with guide *through* stricture. | 3. Wheelhouse. |
| Operation *without* guide to stricture. | 4. Cock. |

Of the nature of these operations it need only be said that they all are external urethrotomies. Nos. 1 and 4 have been already described; No. 2 (Syme) is a perinæal section with the direct object of cutting through the whole length of the stricture, a fine grooved sound being introduced as a guide; and No. 3 (Wheelhouse) has the same object in view, viz. the division of the stricture, but this is attained by passing a specially-formed guide as far as the stricture, opening the healthy urethra at that point, and the parts being retracted deliberate search is made for the entrance to the stricture, down which when found a suitable guide is passed, followed by a fine knife to divide the stricture. It is pre-eminently for those cases where false passages prevent the entrance of the catheter into the stricture; and in whatever case it is used, it presupposes the impassability of the stricture by a sound in the ordinary way.

Whether a stricture calls for operation or not depends on these two points: the time at the surgeon's disposal as determined by the urgency of the case, and the nature of the stricture. When it is determined that operation is required, then the kind of operation that shall be performed will mainly depend on the permeability or not of the stricture by instruments, and also to some extent upon the nature of the stricture. By "urgency" I do not here mean the urgency of retention; but there is a limit to the time that can be allotted to the case, having regard to the patient's general health, state of the kidneys, the presence possibly of cystitis, and many other matters. To say that all strictures can be passed by a catheter, given skill and patience, is going too far; for the clinical question is not one of mere passage of an instrument, but the accomplishment of this within a given time, determined by the necessities of each individual case: and even when the distal end of the stricture is exposed to view, it may not be possible to get even a fine director into and through it. In one such case Wheelhouse's operation failed me. It was that of a man aged fifty-two (April, 1890) who had long suffered from stricture, and a month before I saw him he became "waterlogged" after a drinking bout. A No. ½ catheter is said to have been passed, but from that time nothing could be passed, and when I saw him I performed Wheelhouse's operation. But even with the urethra open I failed to get in the finest guide, and so with the finger in the rectum as guide I cut from behind forwards, completely divided longitudinally the hard cartilage-like stricture, and then passed a No. 6 all round and tied it in.

This is the only occasion in which I remember doing what was to all intents and purposes the Boutonnière operation.

Though the necessity for operating on a stricture is largely determined by the time the surgeon may wait, the nature of stricture must be also considered.

In inflammatory stricture—that form usually attributed to gonorrhœa—the thickening, though in chronic cases organised and fibrous, yet, like the firm, fibrous, subcutaneous growth of a chronic dermatitis, is capable of being largely reabsorbed, and the tissue thereby again made supple. Further, inflammatory strictures are *concentric;* whilst it

is often the *eccentricity* of traumatic strictures that add to their difficulty of treatment. Cross sections through an inflammatory stricture at intervals show the gradually diminishing calibre of the urethra, not caused by intra-urethral change but deposit in the submucosa of erectile tissue, which in proportion to its extent and thickness reduces the size of the canal. As I have already pointed out, the absence of a submucosa in the membranous urethra accounts for its not becoming strictured either by deposit or cicatricial tissue. The reabsorption of the deposit constituting an inflammatory stricture, under treatment, is certain, save in a few extreme cases; but the process is one of cell activity duly excited, and hence, whilst an operation may now and then be necessary for impassability, cartilaginous deposit, or some special condition of this kind, yet unless the operation in some way or other rouses this activity the stricture cannot be cured. Of the four operations already mentioned three of them deal at once with the stricture, and are mainly instrumental in the subsequent cure; but Cock's operation leaves the stricture alone, and is incapable of bringing about a cure, and only to a slight extent aids reabsorption. By the rest that it gives the stricture, local catarrh of the mucous membrane and congestion subside, but I have never known any great amount of dilatation of an old stricture result from mere rest. So-called dilatation by catheters is absolutely necessary after Cock's operation; and in other operations it completes the cure. The idea, however, that a stricture is cured by a dilating pressure or stretching is but half the truth, if so much; and the method, I trust now obsolete, of splitting a stricture is, in my opinion, as ill-judged as it is useless. Certainly for the time the patient is relieved, but in a few weeks he will be as bad as ever again. I have in my mind one case especially who paid a visit to his surgeon to have his stricture "split"—with a specially-devised instrument, with almost the same regularity as that with which he paid his rent.

If stretching or great pressure were requisite for absorption we should not see in dilating a stricture, that where a small catheter is left in, say for four days, it is possible at the end of that time to get in another two or even three sizes

larger. It is the presence of the foreign body that provokes the absorption process, as does that of a thoracic aneurism the absorption of the vertebræ, by direct irritation combined with some pressure. To hurry, therefore, the treatment of a stricture by bougies, whether subsequent to an external operation or not, is not in the long run the best and quickest policy for the patient. The evidence of "irritation" set up as part of the absorbing process is shown in the catarrhal discharge—often very little, but at times abundant—caused by the catheter, and spoken of very inaccurately as due to the "breaking up" of the stricture.

Where the stricture is cicatricial (traumatic) and is severely marked, it generally happens that if it takes four days to dilate it to a four or five size, it will, if the catheter be withdrawn, take about as many hours only to contract again. This is because the stricture is true cicatrix, and behaves presumably like a cicatrix anywhere else; whilst its eccentric contraction often makes the passage tortuous, so that whilst urine can come out even a soft instrument will not permeate it. Such a form of stricture is ill-adapted for treatment by dilatation alone.

In determining which form of urethrotomy shall be done in such a case, the impossibility of curing the stricture by subsequent dilatation would argue against Cock's operation, and be in favour of one of the other forms mentioned which deal directly with the stricture; the one exception to this being the case where though the stricture is not an extreme one, and there is no doubt as to the possibility of subsequent cure by dilatation, yet from previous ill-treatment of the urethra the presence of false passages make it, after patient trial, impossible to enter the stricture. Wheelhouse's operation is here invaluable, if only to find the way in; and that being found, and the incised healthy urethra united—even by suture—the case can be treated from that moment by dilatation.

Though I have implied that it is the structural character of the stricture that determines its curability or the reverse by dilatation, yet in one case, though there was not in the nature of the stricture anything to suggest incurability by this means, I did a Syme, since the stricture was so

painful that attempts to treat it by mere catheterism quite failed. After the operation all went well and the patient was cured in six weeks.

A short account of some of the cases of stricture which have required operation is here given to exemplify the foregoing remarks.

Case I. *Impassable stricture; false passages.*—A patient (March, 1888), aged 22, for a year had micturated in finest stream; at times only in drops. Frequent attempts at catheterism had been made. A month's rest so far improved him that micturition was freer, but I failed to pass any instrument, always apparently getting into a false passage. A "Wheelhouse" exploration revealed three openings in the urethra at the commencement of the stricture; but the finest probe failed to pass through any one of them to the bladder. A Cock's operation was, therefore, performed, and the dense stricture tissue incised longitudinally. Perinæal drainage was adopted for three days, after which a catheter was passed all the way round. He was quite well eight weeks later, passing at intervals a full-sized catheter for himself; but even after many hours' interval there was no appreciable contraction.

This is a very typical case of impassable stricture with false passages. The previous exploration of the urethra was justifiable on the ground that the impassability was due to always entering a false passage; and there was the possibility of cure by dilatation when once the right way was found. This was not so, however, here, and a Cock's operation might, as I have done on other occasions, have been performed at the first. Urethral exploration too would be out of place where, accompanying the other local conditions mentioned, there was severe penile hæmorrhage from recent and perhaps ill-judged attempts at catheterism.

Case II. *Impassable traumatic stricture.*—Man, aged 53 (September, 1885), with eight months' history of traumatic stricture. Water dribbled away, but no catheter could be passed. I explored the urethra as in the last case, dividing the stricture in Wheelhouse's fashion; but I employed for five days perinæal drainage, passing the catheter through the incised stricture, and suturing the healthy urethra ante-

riorly that had been opened. After five days a catheter was passed all the way round, and in seven weeks the patient was perfectly well, passing a catheter at times for himself.

The free incision here adopted was as necessary to obtain a permanent cure of the cicatricial contraction by introducing more tissue, as is tenotomy for a contracted tendo Achillis. Dilatation alone would not have succeeded.

Case III. *Double stricture; one traumatic, the other old inflammatory.*—Man aged 27 (September, 1887). The meatus urinarius contracted down by scars from old venereal sores, and in lower part of bulbous urethra a long hard chronic "cartilaginous" stricture. I divided the cicatricial stricture with a urethrotome, and passing a fine-grooved director through the "cartilaginous" stricture performed Syme's external urethrotomy, passing a large catheter at once all the way round and tying it in. In three weeks the perinæal wound was nearly well, and the patient had learnt to pass a catheter for himself as precaution against recontraction.

It might seem that a small director being passed through the inflammatory stricture (the cicatricial one being overcome), the case might have been treated by dilatation; but this is just one of those cases in which my experience is against trusting to the irritation of a catheter alone to get rid of so much and so hard inflammatory deposit; hence the external urethrotomy.

Case IV. *Old stricture previously dilated; perinæal fistulæ and abscess.*—Patient, aged 42 (May, 1880), had been previously treated by dilatation and was now passing a No. 10 for himself. He applied for relief on account of perinæal fistulæ and an abscess beginning to form behind the scrotum. I performed Cock's operation, opening the back of the membranous urethra—using, however, a sound as a guide—the incision being made to open the abscess and also the fistula. Perinæal drainage was afterwards maintained.

Although efficient drainage of the bladder, either by the perinæal route or by a large catheter passed in the usual way, will usually cause fistulæ to heal, yet it had not been so in this case; and though had I instituted penile drainage the fistula might have closed, yet since the abscess had to be

opened, it appeared the better practice to include the fistula in the incision, and by employing thorough perinæal drainage for a time bring about a more speedy cure.

Case V. *Stricture passable but very painful.*—Patient, aged 36 (October, 1876). This case is the one referred to at the end of my foregoing remarks, as the one of painful stricture for which a Syme was done. After the external urethrotomy was performed on a staff, I passed a catheter through the wound as in a Cock, and, when the patient could bear it, one was passed *per penem*. In six weeks the patient was about, passing a No. 6 catheter for himself.

Case VI. *Old impassable stricture ; retention ; supra-pubic drainage ; subsequently a Wheelhouse operation.*—Patient aged 35 (April, 1889). Two years ago had suffered from retention, relieved by catheterism. For the last year had been gradually getting worse, passing the urine in drops and finally not at all. He was admitted with complete retention for twelve hours. There was no œdema nor extravasation, but a cartilaginous substance was felt in the perinæum. The bladder was punctured supra-pubically and drainage established, the supra-pubic drain being retained for ten days. After this he could relieve himself with great straining by the penis ; and failing after eight days longer in getting any instrument into the stricture I performed Wheelhouse's operation. The stricture being divided on a fine director, a second one was entered immediately behind ; this also was divided and a catheter passed through the wound as in Cock's operation ; seven days later one was passed all the way round. In three weeks the wound had nearly healed, and a fortnight later he was discharged from hospital, passing a full-sized catheter for himself.

This case has been referred to already under "retention of urine," and though he was relieved by supra-pubic puncture, the hard and long stricture barely yielded at all to the rest given it, and I felt warranted in at once doing a urethrotomy instead of wasting time over attempted catheterism ; and the result was as expeditious as it was favourable.

*Conclusions.*

Failing catheterism then, in uncomplicated retention from a *temporary* cause, supra-pubic puncture is the best operation, and it is better done with a fair-sized cannula than an aspirator needle.

In uncomplicated retention from a *chronic* yet remediable cause, puncture with drainage is indicated; and supra-pubic drainage is better than drainage *per rectum;* and any operative interference with the urethra with the mere idea of relieving distension is not advisable, and may be even harmful.

In retention complicated with extravasation, the latter condition it is that must determine the nature of the operation; but retention in cases of extravasation is often not complained of, although much urine may be found in the bladder; the leakage into the tissues probably accounts for this.

In extravasation from stricture it is not enough, nor free from danger of septicism, &c., to merely incise the infiltrated tissues; an efficient drain must be established direct from the bladder. This is best done through the perinæum.

In ruptured urethra extravasation of urine is not necessarily present though the urethra is completely ruptured. Whether present or not union of the torn ends over a catheter is of first importance; and if a perinæal section has to be performed to accomplish this, suture of the urethra is advised, unless there be very much contusion. Where union of the urethra is not feasible from extreme contusion or failure to find the proximal end, a perinæal drain must be established for a time, either through the membranous urethra (Cock's method) or through the proximal urethra; this latter is in most cases preferable.

When a catheter has been passed all the way round it should be retained without changing as long as possible.

In ruptured urethra with retention, with or without extravasation, supra-pubic puncture with drainage may be employed if a catheter cannot be passed, and there is any reason for delaying perinæal exploration. The supra-pubic

opening can be subsequently utilised to find the proximal urethra by "reversed catheterism" in special cases.

Extravasation in ruptured urethra may take the form of a secondary bladder into which the bladder proper empties itself; such a condition presents unusual difficulties in the re-establishment of the normal urethral passage.

All forms of extravasation require the usual incisions to drain the affected tissues, whatever operation is done—perinæal or otherwise—for the establishment of a bladder drain.

In chronic stricture the cases requiring operation are very few compared with those that can be treated by catheterism (dilatation).

In treatment by dilatation of inflammatory stricture, whether with or without previous operation, it is the presence of the bougies causing absorption by irritation, rather than excessive dilatation or pressure, that is required; hence sudden dilatation ("splitting") is condemned.

But, in traumatic stricture, dilatation to the full and long maintained is necessary. Mere "physiological rest" to a stricture as afforded by Cock's operation is very beneficial up to a certain point, but inadequate for cure.

The necessity for operation in stricture cases depends partly upon the time that the patient's condition can allow for palliative treatment, partly upon the nature of the stricture.

In inflammatory stricture, if urethrotomy is determined on, Cock's operation—*i. e.* opening the membranous urethra behind the stricture—is preferable to an operation upon the stricture, except, in certain few very old and hard strictures, and in some cases of stricture complicated by fistula.

In traumatic stricture, if urethrotomy is determined on, an operation that deals with the stricture itself (*e. g.* Syme's or Wheelhouse's) is necessary.

Urinary fistulæ tend to close after the cure of the stricture on which they depend, or if the stream is diverted by operation for a sufficient time. But where in a chronic stricture there is also a fistula opening in perinæo, it will be quicker practice to open up both at one incision, and the cure of the stricture will be that of the fistula.

## DESCRIPTION OF PLATES I, II, III,

### Illustrating Mr. Golding-Bird's paper on Urethral Stricture.

The zincotypes opposite are taken from cross sections of the corpus spongiosum, mounted as microscopic slides, after hardening in chromic acid. With the exception of Fig. 1 they are from a case of chronic stricture, and show the appearances at intervals, from the worst part of the stricture to the dilated urethra behind it.

FIG. 1.—Section of the normal corpus spongiosum for comparison. Observe the truly spongy character of the submucosa.

FIG. 2.—The stricture at its worst. The urethra is a small star-shaped spot in the centre of a mass of hard inflammatory deposit, which has quite filled up the spongy tissue in the neighbourhood. In the original specimen the epithelium is still on the surface of the urethra, thus showing that an inflammatory stricture is a submucous extra-urethral condition.

FIGS. 3, 4, 5, 6, are sections at intervals down the stricture, and show successively a less and less strictured condition, till in the last one (6) the urethra is dilated behind the stricture; some inflammatory deposit is still however seen around the urethra.

The epithelium of the mucous membrane can be seen on these last specimens.

FIG. 1.

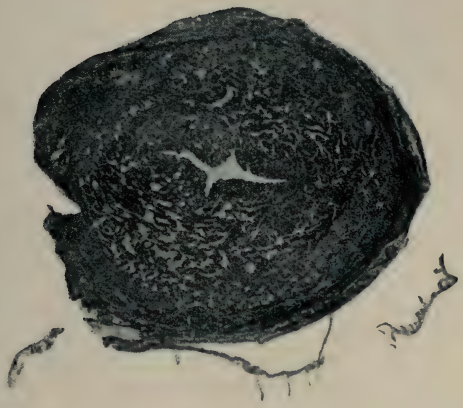
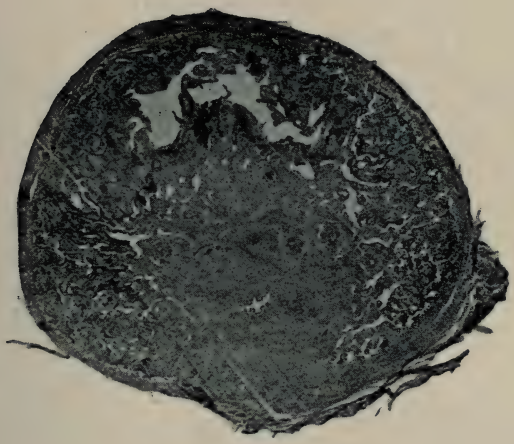


FIG. 2.



*Adlard and Son, Imp.*

FIG. 3.

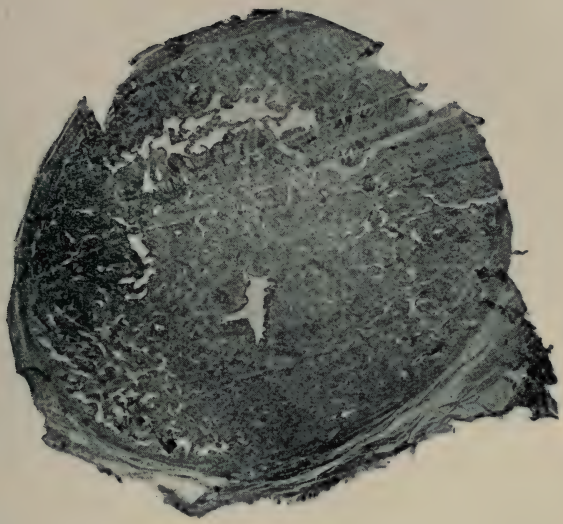
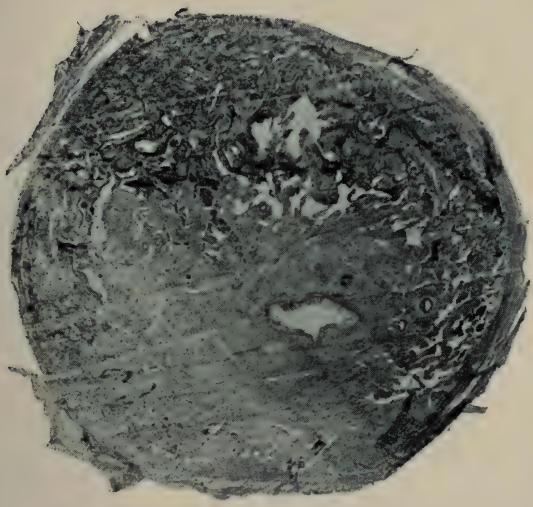


FIG. 4.



*Adlard and Son, Imp.*

FIG. 5.

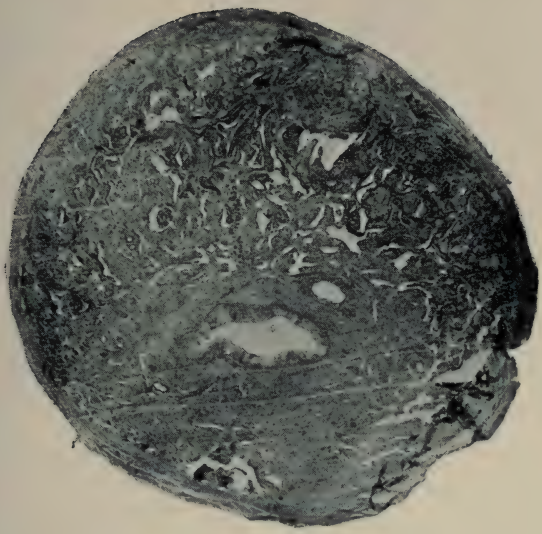
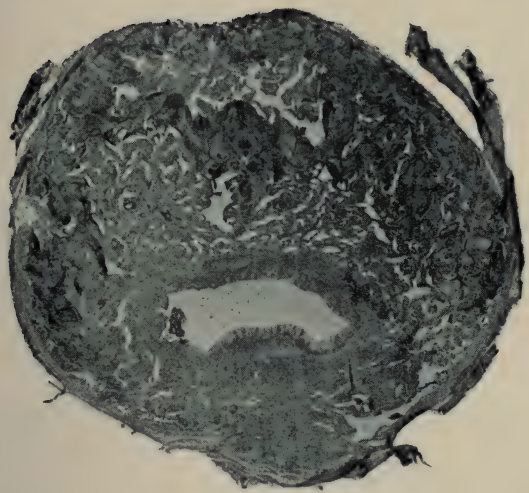


FIG. 6.



*Adlard and Son, Imp.*

# A CASE OF HOMICIDAL STRANGULATION.

By THOMAS STEVENSON, M.D.

The strangulation of an adult person is a rare occurrence, and the homicidal strangulation of an adult is so uncommon that, during the thirty-three years that I have been connected with Guy's Hospital, I have on only one occasion been able to make a necropsy in such a case. Hence the recent death of Mr. Kerwain in the Borough, and the conviction of three men, Waller, Balch, and Noble, of manslaughter (*Reg. v. Waller and others,* C. C. Ct., November, 1892) is of exceptional interest; and the more so as extensive fracture of the larynx was found with no corresponding marks of external injury.

With the exception of one case of fracture of the larynx, met with in the dissecting-room, and placed in the Guy's Museum by Mr. Arbuthnot Lane, we have hitherto had no specimen of this injury in that museum, and I remember only one case of strangulation—and that accidental and non-fatal—admitted into the wards of our hospital.

Mr. William Peter Kerwain was a medical practitioner, æt. 42 years at the time of his death, and resided at Brixton. He had been in practice abroad, and since his return to this country acted as occasional *locum tenens,* and as assistant. He had the reputation of being a steady, abstemious man.

On October 11th, 1892, he was alive and well, but he did not return to his rooms in the evening, as was expected.

Next morning (the 12th), he was seen at 5.30 a.m. in a public-house in the Borough drinking with a prostitute of the lowest class. The two were afterwards seen from time to time in the streets, and in other public-houses; and eventually the woman left him in the company of the three men who decoyed him into a beerhouse. Beer was called for, of which Kerwain drank one glass, although he was already under the influence of drink. The four left the house, and proceeded down the street arm in arm, Kerwain being unsteady in his gait. The three men and their victim entered a dark and narrow passage; and whilst one of the men guarded the entrance the other two effected their purpose. According to his own statement, subsequently made to a fellow-prisoner, Waller throttled Kerwain, by placing the left hand over his mouth and grasping or pressing his victim's throat with the right hand, placing the hand between the shirt-collar and the throat, whilst standing in front of him. Within five minutes of their entry into the passage the three men were observed to run from the other end of it; and immediately after Mr. Kerwain was found doubled up in the passage, and insensible. His collar and sailor-knot tie were tightened, and the shirt-front torn below. The pressure of the collar and tie were relieved, and he was placed on an ambulance. He was thought to be then just alive, but was dead on arrival at the hospital, perhaps five minutes later. When found, froth was exuding from the mouth and nostrils.

When received at the hospital about a quarter of an hour after the deceased had been seen walking in the street, Mr. Carling found the body warm and limp, the face of a leaden hue, and there was a white crescentic depression on the throat, with its concavity upwards, a little below the cricoid cartilage and to the right. The pressure of a collar-stud or of a finger-nail might have produced this mark, which was inconspicuous. There was an abrasion, apparently some hours old, over the second joint of the left middle finger; a recent small bruise below the left knee; and a small abrasion over the left shin.

I inspected the body twenty-four hours after death. Rigor mortis was then well marked, and the body was cold.

There was much cadaveric lividity, more especially about the neck and shoulders. The features were calm, but of a singularly leaden and ashy hue. The ears and lips were intensely livid. The upper part of the forehead and the top of the bald scalp were covered with minute dusky spots of ecchymosis, varying in size from that of a pin's head to about four times this size. The eyelids were nearly closed, and the pupils were of the natural size. The crescentic mark on the neck had assumed a dark purple hue. Nowhere else on the neck was there any ruffling of the cuticle or sign of violence. The hands were semi-clenched.

The post-mortem examination was made forty-eight hours after death by Mr. Carling in my presence. The external appearances were those already described. There was no fracture of the skull nor extravasation of blood beneath the scalp, and no sub-conjunctival ecchymoses. The tongue was in contact with the teeth, but not indented. There was much dark blood effused into the tissues and muscles of the neck from the hyoid bone downwards to near the top of the sternum, around the larynx and trachea. The larynx was extensively fractured, and the hyoid bone also. On the right side the thyroid cartilage had its superior cornu broken completely off from the body of the cartilage; and on the left the cartilage was broken across about one eighth of an inch from the superior cornu. A vertical fracture extended from the anterior notch downwards and a little backwards to the lower border of the cartilage. The cricoid cartilage was fractured, the fracture being on the right and towards the front, and the two ends of the fractured cartilage overlapped each other. The hyoid bone was broken across just behind the right lesser cornu.

The epiglottis was raised; the glottis was free from obstruction; there was no blood, food, or obstruction in the air-passages. The trachea was much injected. A small quantity of the semi-fluid stomach-contents had regurgitated into the lower front of the œsophagus. There was no marked hyperæmia of the tongue. The lungs presented on their surface the silvery patches of emphysema described by Tardieu; there was some effusion of blood into the air-cells, and a couple of small apoplectic nodules of effused blood in

the substance of the left lung. The heart showed no unusual engorgement and was contracted; the right ventricle contained a little dark blood-clot, the left was empty. There was an effusion of blood beneath the pericardium over the left ventricle, one inch in length by one third in breadth. The brain presented no unusual appearances. The kidneys were somewhat hyperæmic. There were no other noteworthy post-mortem appearances in the body.

There can be no doubt that the unfortunate man was seized by the throat in the manner already indicated, and his larynx crushed. Insensibility would probably immediately result, and Kerwain would be unable to cry out, or in his helpless condition to make any effective resistance. Here it may be observed that the stomach of the deceased contained three-quarters of a pint of almost entirely liquid contents, containing about $6\frac{1}{4}$ per cent. by volume of alcohol. As average public-house beer contains about $5\frac{1}{4}$ per cent. only of alcohol, it is clear that something stronger than beer had been drunk by the deceased. A minute analysis of the stomach-contents and of the various viscera of the body negatived the supposition that Kerwain had been drugged with a narcotic.

In conclusion, I may remark that the post-mortem appearances in this case are in accord with those described by Tardieu as met with by him in cases of strangulation.

# THE

# REGENERATION OF FIBROUS TISSUE,

AS

# EXEMPLIFIED IN THE UNION OF TENDON.

By A. H. TUBBY, M.S.

The question of the precise origin of the ultimate components of new fibrous tissue has recently received an additional stimulus from the very able observations of Messrs. Sherrington and Ballance, detailed in the 'Journal of Physiology' for October, 1889. These observers determined to repeat Ziegler's classical experiments with the view of deciding as far as possible the respective merits of those authorities, who on the one hand claim that in the leucocyte we have the beginning and the end of the process; and of their opponents, who assert that the leucocytes play an entirely subordinate *rôle*, and that the chief actor is the "plasma" cell.

In the course of a series of observations on the processes by which tendons unite, extending over thirteen months, I strove to ascertain how far in this particular variety of connective tissue, after injury, the appearances were in accord with the views of the Cohnheim-Ziegler school; or militated against them, and supported those of more recent observers. And in passing let me express my indebtedness

to Messrs. Sherrington and Ballance for their most admirable article, and for the guidance they have afforded me in following the complexities of the matter at issue.

In normal tendon there are, as is well known, white connective-tissue fibres and tendon cells. Of the latter, recent observations show that there are three varieties, the large "fixed" cell, seen as an oblong body in sections parallel to the long axis of the tendon, and in transverse sections as of irregular outline, with processes extending into the ramifications of the connective-tissue space, and generally uninucleated; also migratory cells are found, which wander freely through the interfascicular spaces. Chief among the latter are the "plasma" cells of Waldeyer—oval or slightly irregular in outline, occasionally showing one or more processes, larger than the leucocyte, and with a large oval nucleus and granular cell-substance, several cells often forming clumps or aggregations. The other kind of wandering cell is practically indistinguishable from the leucocyte except by special stains.[1]

Now my observations support the view that it is in the plasma cell we must seek the origin of permanent cicatricial tissue.

In specimens of normal tendon but very few round cells are seen either between the fibres or the bundles. On dividing the tendon it is soon apparent that the number of cells is immensely increased, and that these form clumps between the bundles, and then between the fibres, which push the latter apart, and give rise to a frayed-out appearance of the divided end. These masses of cells are probably the latent cells or 'schlummernde Zellen' of H. Schmidt,[2] described by him as becoming visible only under the effect of prolonged stimuli such as the effects of injury. But more on this point later.

*Method of experiment.*—The tendo Achillis in full-grown rabbits was divided from the outer side, as it is easier to avoid wounding the artery, an accident likely to occur in dipping the knife when introduced in the classical way.

[1] Cf. 'Proc. Roy. Soc.,' No. 317, Kanthack and Hardy, "On the Characters and Behaviour of the Wandering (Migratory) Cells of the Frog," &c.

[2] Virchow's 'Archiv,' Bd. cxxvii, S. 96, and Bd. cxxviii, S. 96.

The puncture was closed by a solution of iodoform in collodion, and a strip of sublimate gauze bound round the limb just above the os calcis. No attempt was made to fix the limb by plaster-of-Paris or other supports, as it was felt that any such effort would be fruitless in the case of rabbits; they would probably attempt to digest most materials. The only precaution adopted was to minimise the space allotted to the animal, and so prevent its moving about freely. The rabbits were then left for the following periods: three, seven, fourteen days, four, five, seven, eight, nine, thirteen, and thirty-three weeks, and the longest period, thirteen months.

For microscopical observation, the tendon with its sheath entire as far as possible, was removed, and placed at once in weak Flemming's fluid of chromo-aceto-osmic acid for twenty-four hours. By immersion in this it became sufficiently hard for cutting. Some portions were then stained *en masse* in a mixture of alum-carmine and osmic acid[1] (Zoltán y Roboz' formula), and recommended by Mr. Bolles Lee. This stain has a very direct selective action on protoplasm; active chromatin is stained red, while resting chromatin is stained purple, the rest of the cell brown. I can strongly recommend this solution for giving the cells clear definition, and distinguishing between the active plasma cell and the sluggish leucocyte. The tendon pieces were then embedded, some of them in paraffin, and others in celloidin. In other instances, after using the fixation fluid, the masses were embedded at once in celloidin, cut, and stained individually in hæmatoxylin and eosin, at the instance of my friend Dr. Beneke of Brunswick.

It has been inferred that it is almost impossible to embed tendon in paraffin to yield satisfactory sections, since the fibrous tissue hardens and becomes brittle with the heat of the bath. But acting on the advice contained in a letter from Mr. Bolles Lee, I avoided using xylol, and employed cedar oil for clearing, taking care to keep the pieces of tendon at least three days in the oil; and then soaking in paraffin at a temperature of 48° C., and not more than thirty to forty-five minutes. In this way I succeeded in procuring good results.

[1] Bolles Lee, 'Microtomist's Vade-Mecum,' second edit., p. 85.

Experiment 1. *Division of the tendo Achillis from the outside; rabbit killed immediately afterwards; microscopical appearances.*—The ends of the tendon are seen to be cleanly cut and square, while the individual fibres are parallel. The tendon stump is embedded in a complete cap of effused blood-corpuscles. A few white blood-cells are visible, and are as a rule rounded and contain but one nucleus. At places the tendon bundles are separated slightly from one another, and in the intervals some red blood-corpuscles are seen; in addition a few rounded cells, very doubtfully white blood-corpuscles, are visible creeping into the extremities of the divided tendon. No blood-vessels are demonstrable between the fasciculi, nor are there any aggregations of cells in the interfascicular channels. It should be stated that there were a few drops of blood at the time of operation, and some was found effused in the subcutaneous tissue.

Experiment 2. *Three days after division; appearances.*—The tendon stumps show at their extremities considerable alterations, inasmuch as they are rapidly becoming infiltrated with large cells—plasma cells—which separate the individual fibres and obscure them. No red blood-discs are perceptible between the tendon fibres. The effusion between the tendon stumps consists very largely of numerous red blood-discs whose outlines are rapidly becoming ill-defined. Fibrin fibrils are observable amongst the discs. In the effusion are seen numerous longitudinal vacuolations, some with ill-defined, and some with clean-cut margins. A few vacuolations are surrounded by an almost continuous layer of plasma cells with well-defined nuclei (vasoblasts), the general impression given being that of a branching capillary vessel, but lacking the flattened endothelial lining. Occasionally the vacuolations are seen to branch. But the most striking point about the effusion is the large number of plasma cells, some apparently dividing. Many of them have lost their rounded outline and become oval, while a few are fusiform or irregular in shape. Leucocytes are still abundant. The fixed tendon cells are not greatly changed. In some cases they appear less oblong and more flattened near the site of operation, but this is owing per-

haps to variation in the angle of section at different parts of the tendon. The nucleus is found more towards the centre of the cell and is less rounded than normally. Certain uninucleated cells, latent cells, or 'schlummende Zellen' of H. Schmidt, smaller at this stage than plasma cells or leucocytes, are evident between the bundles of fibres and fibres themselves. These are not visible in normal tendon.

Experiment 3. *Seven days after division.*—The sharply-cut appearance of the tendon stumps, so well marked soon after division, has quite disappeared. The ends are now ragged, and several isolated masses of tendon substance are seen surrounded by what is apparently fibrin. Just above and below the point of section, between the individual fibres, there are everywhere seen numerous large plasma cells, often collected in groups, and undergoing rapid division, occasionally bipolar in shape. Small round cells, leucocytes, are by no means so abundant in the reparative material, and by this time nearly all trace of the red corpuscle has disappeared. Plasma cells are seen grouped together in large islets, and are surrounded and supported by a faintly staining, somewhat homogeneous or hyaline material. In some sections a well-defined mass of deeply stained uninucleated cells is visible. Many of these are rounded, others show one slight prominence. In addition there are larger, faintly stained, oval, nucleated cells. It is probable that at this position masses of plasma cells are forming in the interfibrinous network, and are increasing at the expense of the leucocytes, as described by Messrs. Sherrington and Ballance.[1] The vacuolations seen on the third day have quite disappeared, and at various parts linear arrangements of large, rounded, granular cells, often ending in clump-like masses of similar cells, are evident (see Photo-micrograph No. 1). I am disposed to regard these cells as vasoblasts, and the arrangement as preliminary to the formation of new vessels in the uniting material (see Photo-micrograph No. 2). The clump-like

[1] 'Journal of Physiology,' vol. x, p. 571.

appearance is due to the sudden twisting of the procession of cells; and afterwards of vessels which course freely and in many dippings through the new tissue before becoming connected, on the one hand, with the muscle above, and, on the other, with the vessels derived from the periosteum to the stump of the tendon below.

In connection with this experiment, the presence of large plasma cells between the tendon fibres and bundles should be noticed. They are apparently directly derived from the clumps of very small nucleated cells noticed in Experiment 2. They have now assumed the appearance of plasma cells, and, what is more to the point, are arranging themselves in line between the fibres at a short distance from the point of section. But near the site of operation they are sometimes directly continuous with small vessels whose walls are composed of a single layer of oval and rounded cells. It is highly probable that in the 'schlummende zellen' we have the antecedents of vasoblasts which form communicating channels between the muscle above and the uniting material below. Until this connection occurs the scar tissue receives much of its supply, in the first place, from lymph derived from the hypertrophied vessels found in the tendon sheath; and, later, anastomosing vessels form across the scar tissue from side to side. The well-defined outline of the fixed cells is obscured, and they tail off occasionally, but far from universally. The nucleus still remains of normal size and shape.

Experiment 4. *Fourteen days after division.*—Referring for a moment to the structure of the thickened sheath: the blood-vessels are not so numerous as on the seventh day, and are much less dilated. The entire thickness of the sheath, at the point of section of the tendon is composed of a ground substance of ill-defined fibres running in all directions and feebly stained. Many varieties of cells are seen (see Photo-micrograph No. 3). There are a large number of overgrown granular cells, some of them nearly twice the size of white blood-cells, and exhibiting infinite variations in shape. Some are oval, some have one, two, or more processes,

and others are considerably elongated like short varicose fibres. The latter have two or more nuclei, and frequently blend with the indistinct fibrous ground substance. But the more the cells have advanced towards the fibrous tissue stage, the greater difficulty is experienced in finding the nuclei; and in some instances the latter appear to be entirely absent. A noticeable feature in the tendon sheath, as compared with that in the two previous experiments, is the continued diminution of leucocytes.

The ends of tendon have now completely lost all form and shape, and are blending imperceptibly with the scar tissue, *i. e.* the latter is dove-tailing into the old tendon. In this the interfibrous and interfascicular cells are much increased in number and prominence, and their arrangement in linear series is evident at a greater distance from the point of section. In the fixed tendon cells no further change can be detected except that the nucleus is more often seen in the middle of the cell. Islands of plasma cells are visible, and the cells are elongating rapidly. Leucocytes are still seen in moderate number in the centre of the scar, but at its periphery they are few and scattered; it is possible that some may have wandered out from the new vessels. The young fibres are more defined, and exhibit an inclination to arrange themselves parallel with the long axis of the tendon. This appearance is only seen near to the tendon itself, but at the furthest point from the ends there is only a confused mass of faintly fibrous ground substance, islets of plasma cells, and leucocytes, such as may be demonstrated throughout the plastic material on the third day. It would appear in tracing the formation of permanent fibres that fibrin-filaments have no part therein, since all trace of them disappears before this date, and they give way to a filamentous ground work formed by the processes of the plasma cells. Amongst the latter some giant-cells are visible, with four, six, eight or more nuclei.

Experiment 5. *One month after section.*—Running down into the new tissue from the tendon are seen beautifully looped vessels, and it is now possible to demonstrate the direct continuity of vessels from muscle to old tendon and

thence into scar, forming anastomotic chains. This is especially the case where the rounded ends of the old tendon are traceable. New vessels skirt the margin closely. No leucocytes are visible in the tendon itself; while now that blood-vessels have formed, the 'schlummende Zellen' and their derivatives are distinctly less in evidence. In the line of union all traces of leucocytes have gone; the processes of the plasma cells near to the tendon are insinuating themselves in all directions between the old fibres.

Experiment 6. *Five weeks after division.*—Considerable hæmorrhage took place at the time of operation, and the animal subsequently wasted and died. Indeed, it is very noticeable that within three or four days after the administration of ether to rabbits, rapid wasting sets in and persists for several weeks. Recovery, however, takes place in nearly all cases.

In this case the microscope showed very conclusively that the regenerative changes were more advanced in the proximal than in the distal portion of the cicatrix, owing doubtless to the greater pabulum in early stages due to the larger quantity of blood coming down from the muscle. It should be stated that as the interfascicular and interfibrous development of vasoblasts is, in the case of the tendo Achillis, much greater near to the muscle than at a distance, firmer union thus taking place near the muscle than at a greater distance, it seems to afford us this practical hint. If we wish to obtain rapid union with as little elongation of the tendon as possible, divide near the muscle; if the reverse, at a considerable distance.

Experiment 7. *Seven weeks after division.*—The reparative substance is showing in its proximal part very distinct wavy fibrillation. Sections from this tendon give further support to the view that the fibres arise from the plasma cell. All stages of these are traceable in previous specimens from the round to the oval, and thence to the fusiform nucleated cell. In a section taken from a tendon at this date the nucleus is seen to be fusiform in shape; other spindle-shaped cells have lost their nuclei, and are to all

intents short fibres. The number of vessels is much fewer in the scar, and round cells are generally absent.

In another rabbit operated on the same date, and which died eight weeks afterwards, considerable suppuration occurred. The appearances here were somewhat different. The vessels in the scar were more numerous and more thickly walled, and the new fibrous tissue is mainly deposited around the vessels, the interstices being occupied by plasma cells in all stages. It is curious to note in this suppurating tendon certain degenerating round phantom cells with three nuclei, which are probably leucocytes or pus-cells.

Experiment 8. *After thirteen weeks.*—The chief point noticeable here is that the young fibres have become more parallel in the proximal part of the scar, but the arrangement is still a little indefinite, and lacks the regularity proper to normal tendon. The process of disappearance of blood-vessels is capable of being observed at a point more distant from the tendon stump. The walls lose their sharp definition, while the lumen is occupied by a number of small round cells, giving rise to circumscribed masses. The nuclei of the fixed tendon cells have now divided, and the bodies of the cells are broader and flatter, while the ends tail off. But there is no evidence to show that they are direct agents in the formation of young fibrous tissue.

Experiment 9. *After thirty-three weeks.*—The uniting material now more closely assimilates the appearance of normal tendon, and the young fibres are more regularly arranged; the oval and fusiform cells are more or less in procession. But the following differences are noticeable. The fibres are by no means strictly parallel; the spaces between them are much greater than normal; the individual fibres branch; and some of the new cells resemble the fixed cells of the tendon stump, in their disposition between the cells, their more geometrical outline, and the presence of a single round nucleus. The nests of cells (now probably occluded blood-vessels) are very much smaller, while the included cells are considerably altered from the round to the fusiform shape. In fact, the outer zone of nest-cells is forming fibrous tissue.

Experiment 10. *Thirteen months after division.*—Here it is interesting to note that although there was sufficient elongation of the uniting material to render the muscle of little use, yet there are no evidences of degeneration of the muscular fibres. Sections show, too, that over the greater part of the uniting material the appearance of normal tendon is not quite regained. While many of the new fibres are parallel, some wander freely, and there is still a looseness of pattern. In addition small blood-vessels are seen in the scar. The vessels, however, are becoming gradually occluded; this is evidenced by injection from the main artery of the limb. Transformation into perfect fibrous tissue is still delayed at the distal end of the tendon. Many large oval granular cells are seen in the latter position, some of which are tailing; while others are more advanced towards the spindle shape. Some of the cells, gradually seen to be derived from the plasma cells, now very closely resemble fixed tendon cells. Hence we may venture to conclude that perfect regeneration is possible, but that it is far from complete thirteen months after division; and by traction considerable stretching of the uniting material may occur.

*Remarks.*—The microscopical appearances detailed above are quite in accord with the view that in the plasma cell we have the immediate precursor of the permanent uniting material. Since Waldeyer[1] in 1874 described them as large round cells containing large granules, Ehrlich[2] has in 1877 and subsequent years more fully dealt with the question, and has differentiated from them certain other cells in the same tissues, which resemble in some points Waldeyer's plasma cells, especially in containing large granules, but differ from them in their staining reactions. Especially so is this the case, if they are stained with anilin colours and after-treatment with alcohol are adopted. The Mastzellen of Ehrlich show the nucleus and cytoplasm generally unstained, but the characteristic granules in the cytoplasm very deeply tinctured. Kanthack and Hardy,[3] however, in the most

[1] 'Archiv f. mik. Anat.,' Bd. xi.

[2] Ibid., Bd. xiii, and Reichert and Du Bois-Reymond's 'Archiv,' 1874, p. 166.

[3] Loc. supra cit., p. 268.

recent work dealing with this question, classify the cells found in blood and lymph into eosinophile or non-phagocytic cells, hyaline or phagocytic cells, and, in connective tissues, basophile cells. They affirm that under the influence of various abnormal stimuli a fusion of eosinophile with hyaline cells occurs, and that the latter cells form a distinct plasmodium.

The obstinate nature of the material with which I have worked, its brittleness, and the difficulty of obtaining sufficiently thin and evenly stained sections, have forced me to be content with distinguishing merely between plasma-cells, in Waldeyer's meaning, and leucocytes. As defining the two, I have taken those cells which are the smaller, and nearly uniformly round, with occasional division of the nucleus and absence of any subsequent change of form, to be leucocytic; and on tracing such cells through various sections their gradual disappearance and replacement by the other variety of wandering cell have been further means of distinction. The plasma cells are known by their large size, great irregularity of form, large nucleus, and granular protoplasm, somewhat scanty in comparison with the size of the nucleus; they are not seldom found collected into clumps, and here the various stages of tissue-formation may be traced. In normal tendon they are not easy to demonstrate, and I have not succeeded in doing so satisfactorily, but after division they were found on the third day. They were doubtless present earlier. Sherrington and Ballance[1] note their appearance in eighteen hours in the glass chambers inserted by them in the peritoneal cavity.

On the third day some of the plasma cells have become vasoblastic in function. They are forming very distinct boundaries to certain longitudinal vacuolations in the coagulum, and from that time forwards can be traced till they assume a closely united single layer of spheroidal or columnar cells composing the vessel wall (cf. Photo-micrograph No. 2). Still later, when the vessels are becoming obliterated by a proliferation of round cells, the vasoblasts are giving rise to young fibrous tissue in the thirteenth week. There can be no doubt that the plasma cells too

[1] Loc. supra cit.

are distinctly fibroblastic, the great majority of them being so from the first, while the minority pass through a vasoblastic stage of existence. In either case, by experiments extending over a sufficiently prolonged period, all the stages of formation of fibres from these cells are made evident. The nucleus of the cells remains rounded for a very considerable period, but in twelve or thirteen weeks it is often fusiform, and in other cases it has disappeared altogether at this date. Such development of fibrous tissue is always best marked in the immediate neighbourhood of the vessels, and it is not till the intervascular cells are distinctly fusiform that the vessels become blocked.

In the union of tendon the grouping of the plasma cells into islets is well exemplified, and follows on in point of time and place the clumping together of leucocytes (vide Experiment 3). In these clumps there are not seldom seen giant-cells with several nuclei; and such cells are probably due, as Sherrington and Ballance have shown, to the presence of ingested leucocytes. The clumps of plasma cells forming distinct islets in the earlier experiments should be distinguished from other clumps and groups occurring about the twelfth or thirteenth week, and due to obliteration of the vessels. At first the latter become filled with round migratory cells; these are replaced by plasma cells, and the latter then become converted into fibrous tissue. From my observations it seems that in the gap between the tendon stumps the plasma cells, in addition to forming fibres, have another rôle. From them arise cells closely similar to, if not identical with, the fixed tendon cells.

The part played by the fixed tendon cells in regeneration of the tendon is very subsidiary. For a long time it is difficult to be sure that any change occurs in them, but as time progresses the nuclei are seen at first in the centre of the cell, and then divide, with some tailing off of the cell protoplasm at either end. In any case the cell processes so formed are insignificant, and do not attain any length. During the period of my observations no distinct fibres could be traced arising from the oblong tendon cells.

Leucocytes, or, to be more precise, round non-phagocytic cells, play, as might be expected, but a small part. They are

at first exceedingly abundant but subsequently they are entirely replaced by the plasma cells. In fact, the former serve merely as an nidus for and a pabulum during the development of the latter. Where the process of union is most advanced, there the absence of the leucocytes is most marked; this is especially so at the proximal portion of the new tissue. They give rise to neither vessels nor fibres.

The dovetailing of old and new tendon material observed both by Paget and Adams is brought about as follows:—In the first place we have after section the appearance of the "latent" or 'schlummende Zellen' of H. Schmidt as rounded cells of small size and in groups. They are replaced by larger cells, similar to plasma cells, which form vasoblasts and fibroblasts. In addition there is an increase of plasma cells preceded by leucocytes from the effused new material. This increase occurs just at the site of section, while the development of the latent cells can be seen at a considerable distance—half an inch or more—from the point of operation, with, frequently, at first a clear interval between them and the ingrowing cells derived from the uniting material.[1]

The disappearance of the red corpuscles is in rapid progress on the third day, and by the seventh day nearly all trace of them is lost. But it is questionable how far the vacuolations mentioned above correspond to the space previously occupied by red corpuscles. It is more than probable that the plasma cells dispose by phagocytosis of the blood-discs within a given area, and then take upon themselves a vasoblastic function. From time to time vacuolations form which are not surrounded by plasma cells. Such vacuolations are seen in the scar as late as the thirteenth week, and may be due to the formation of fat, and its absorption.

Fibrin filaments seen in the early stages, and up to the seventh day, disappear with the advance of the plasma cells, and form no part of the young fibres, which clearly originate from the plasma-cell processes.

[1] This interval becomes bridged over by the conjunction of the two sets of cells.

## PHOTO-MICROGRAPHS,

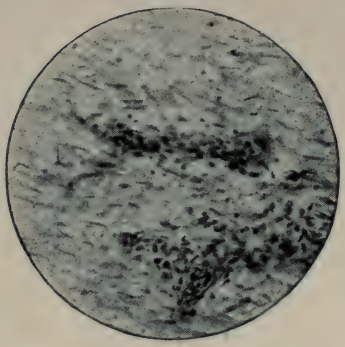
### To illustrate Mr. Tubby's Article on "The Regeneration of Fibrous Tissue."

No. 1.—Shows collections of plasma cells (vasoblasts) in uniting material, forming the boundaries of branching vacuolated spaces (p. 113).

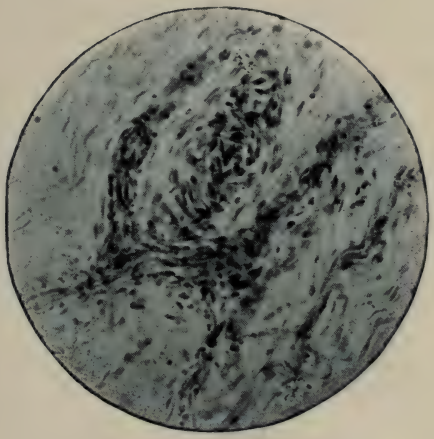
No. 2.—A more advanced condition of No. 1. Shows new vessels, nearly formed, in uniting material (pp. 113 and 119).*

No. 3.—Shows collections of plasma cells, many of which are rapidly elongating. Giant cells are also present (p. 114).

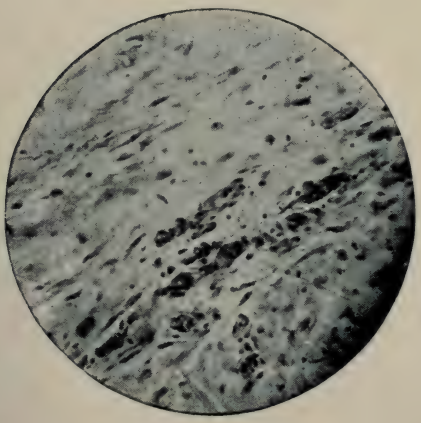
No. 1.



No. 2.



No. 3.



*Adlard and Son, Imp.*

# SOME CASES OF MALIGNANT DISEASE OF THE CHEST AND ABDOMEN.

By FREDERICK TAYLOR, M.D.

Probably very little can be added to our knowledge of the general behaviour of malignant disease, looked at from a clinical point of view, in reference to its invasion of various organs, the symptoms it produces, and the means by which it may be diagnosed. But in a complicated structure like the human body the possibilities are so numerous, and the details even in similar cases may be so different, that even now there may easily arise from time to time cases which present features of considerable interest both clinically and pathologically. I should have hesitated—indeed, I have delayed for a long time the publication of any such case alone, but having accumulated five instances, which are somewhat different from the cases of cancer as we know it invading the stomach, liver, lung, or œsophagus, I have ventured to report them in this article, hoping they may be found of interest to the readers of this volume. The accounts have been considerably abridged from the reports of

the clinical clerks; otherwise they would, I fear, have been found intolerably wearisome; and I have added to each case some remarks upon the points of importance which it seems to me to present. The cases are as follows:

1. Perforation of the aorta caused by epithelioma of the œsophagus; death from hæmorrhage.
2. Suppurating cancer of the lung simulating empyema; incision and drainage with temporary relief; death from hæmorrhage.
3. Multiple carcinoma of the spine and ribs, secondary to scirrhous carcinoma of the breast.
4. Cancer of the right kidney and spine; small (? atrophied) pancreas; deficiency of colouring matter in the fæces.
5. Cancer of the kidney, ureter, and bladder; hæmorrhage from the bladder.

Case 1. *Perforation of the aorta caused by epithelioma of the œsophagus; death from hæmorrhage.* (From notes by Mr. W. Lansdale.)—Charles L—, a publican, æt. 55, was admitted in a state of severe collapse on April 7th, 1885.

For some time previous to two months ago the patient had been very subject to cramps in the lower extremities, and sometimes in the trunk. At the time mentioned he began to have attacks of violent retching, when he brought up only frothy mucus without any admixture of blood. Each of these attacks lasted a day or two, and then he felt much relieved, being quite free from any feeling of illness in the interval between the attacks. About two months ago also he began to have severe pain in the epigastric region, and vomited after taking food. He also had a cough, and blood was noticed in his sputum. He never had any fever, and there is no history of rheumatic fever or of gout.

Epigastric pain, inability to retain his food, and cough with bloody expectoration have been his chief symptoms, and have got worse. Immediately after swallowing any solid food there has been severe pain at the pit of the stomach, followed by retching. Sometimes the food seemed to "stick halfway down;" then he would vomit, cough, and expectorate a number of small blood-clots, the coughing being

accompanied with pain at the epigastrium. Latterly the quantity of blood expectorated has much increased. The last few days his voice has been hoarse.

*Present condition.*—He lies on his right side. He is dark-complexioned, blanched, dusky-looking. His voice is thick and rather hoarse, and his cough is somewhat ringing. He is too ill to be examined thoroughly, but on the right side of the chest there is supplementary breathing; whereas the left side though giving moderately good resonance, presents marked deficiency of vesicular murmur, deficient tactile vibration, and loss of vocal resonance at the lower parts. There is some inspiratory stridor in the effort to cough. He is almost pulseless; examination of the heart reveals no murmur. He is too ill to make a laryngeal examination. I diagnosed aneurism of the aorta compressing the left bronchus obstructing the œsophagus, and probably pressing on the left recurrent laryngeal nerve.

He was ordered 5 grains of carbonate of ammonia every three hours. He became worse during the course of the afternoon, and died in the evening.

The post-mortem was made by Dr. Hale White. Features sunken, body much wasted. Larynx and trachea normal. Both pleuræ and the right lung healthy; left lung tough, airless, and bluish in colour. The posterior surface of the left bronchus for about an inch and a half bulged inwards; the bulging began half an inch from the bifurcation; it did not by any means occlude the bronchus, but diminished its calibre considerably. It had not ulcerated, and the bronchial mucous membrane was intact.

Heart 13 oz., normal. At the lower surface of the arch of the aorta, exactly opposite the left subclavian artery, was a round hole about the size of a pea; through this a part of a malignant tumour of the œsophagus had grown, and presented quite a shreddy filament free in the aorta. This was long enough to reach across the aorta, and probably by friction caused the ulcer opposite the hole. This ulcer was about the size of a sixpence, rough, shallow, and irregular. The shreddy piece of growth had not whipped out any fibrin; it

did not quite fill up the hole in the aorta, so that leakage of blood must have taken place.

From a point two and a half inches from the cardiac end, upwards for four or five inches, the whole of the circumference of the œsophagus was infiltrated with malignant growth. The interior was darkish brown from altered blood, and was very irregular. It compressed the bronchus, and opened into the aorta as already described. The left recurrent laryngeal nerve was pressed upon.

The stomach contained 23 oz. of recent blood-clot. The liver weighed 58 oz., was cirrhotic, pale yellow, tough, hard, not hobnailed. The gall-bladder contained facetted gall-stones. The intestines were normal. A gland near the diaphragm was large and hard from the presence of malignant growth. The kidneys weighed 13 oz., and were normal.

*Remarks.*—The above case is an instance of the not very common occurrence of the invasion of arterial walls by cancer. It is well known that veins are more liable to be attacked by cancer than are arteries. The large arteries not unfrequently pass absolutely untouched through the same tumour, which grows into the veins and obliterates them by projection of large nodules into their calibre; so that when it becomes a question whether a mediastinal tumour, recognised by physical signs to be present in the chest, is an aneurism or a cancer, the fact that the arteries or veins are much interfered with may give valuable aid in the decision. Case 5 illustrates the growth of malignant tumours into the veins, and the exemption of arteries. But occasionally arteries are attacked. Drs. Wilks and Moxon[1] state that this takes place especially in the case of the thinner wall of the pulmonary artery, but "even then we have never seen the elastic coats changing to cancer like other tissues, but they are pressed in and wasted away by the cancer, and so a cancerous nodule can be seen projecting into the interior." "When, however," they go on to say, "a large artery is near a cancerous sore in a sloughing state the vessel's wall is apt to give way along with other tissue in the sloughing process. This generally results from sloughing epithelial cancers.

[1] 'Pathological Anatomy,' 2nd ed., 1875, p. 162.

Thus such cancers in the pharynx or œsophagus may reach the carotid; a cancer of the œsophagus only may reach the aorta." The present case, no doubt, falls within the above description. The position and relation of œsophageal cancer render it very liable to slough; and though the term slough is not used in the description of the post-mortem, the irregular brown surface of the growth, the shreddy filament presenting in the aorta, and the fact that the blood from the aorta had escaped into the œsophagus, are indications that the growth of the cancer was being complicated by degenerative processes.

The circumstances under which the patient came to our notice gave little opportunity for accurate, much less for prolonged, clinical observation.

The history and the physical signs, so far as they could be ascertained, were compatible with aneurism of the aorta. The hoarse voice and somewhat ringing cough were explained by compression of the left recurrent laryngeal nerve, the absence of respiratory murmur on the left side by compression of the left bronchus, and the dysphagia by compression of the œsophagus; and all of these are frequently caused by aortic aneurism. It was an obvious suggestion that the pallor and the collapse rapidly becoming fatal were due to rupture of the vessel wall and extravasation of blood into the œsophagus, upon which the aneurism had pressed. It is, however, only another instance of the similarity between symptoms, produced by tumours of whatever kind, arising in the limited space of the mediastinum among structures so important, that enlargement in any direction is bound to be followed by interference with the right of way of food, air, blood, or nerve-impulses. Looking back at the history, we might think that the short duration of the symptoms and perhaps the predominance of sickness and dysphagia could have justified fuller consideration being given to œsophageal cancer. But even then epigastric pain, cramps in the trunk and lower extremities, cough, and expectoration of blood are quite as likely to be due to aneurism as to œsophageal epithelioma. Only the condition of the aperture in the aorta with its loosely fitting shred of new tissue necessitating leakage, as Dr. Hale White remarks, makes it possible

that the expectorated blood came in part, if not entirely, from the œsophagus, and not from the lung.

It will be readily allowed that no treatment was of any avail when he came under our care. His pallid appearance and collapsed condition show that the large hæmorrhage found post mortem had already taken place, although none of the blood was rejected by vomiting, or had escaped by the lower bowel and rectum.

In 'Virchow's Archiv' (1891, Bd. cxxvi, p. 407) is an article by Dr. Reinhold Altmann on "Perforation of the Thoracic Aorta from the Œsophagus." He reports that out of 6886 inspections conducted at the Pathological Institute of Breslau in the course of ten years, 38 deaths were due to spontaneous bleeding from the aorta or its branches. In 14 cases the walls of the artery were primarily diseased; in 24 the artery was invaded by a diseased process starting in the neighbourhood. In 12 of this last group, the arterial wall was involved in suppuration, and in the remaining 12 it was destroyed by a cancerous growth. In 4 of these the aorta was the artery involved, and Altmann gives accounts of the post-mortem inspections. The comparative rarity of the occurrence is thus shown, since it happened only four times in 6886 cases, or once in 1741 cases—a percentage of ·058. In our own hospital this would mean at the present time about once in three to three and a half years. Another cause of perforation of the aorta from the œsophagus, of which Altmann records an instance, is the impaction of a foreign body in the gullet, such as a coin or piece of bone.

In a previous number of the same journal[1] Zahn records two cases of perforation of the aorta by œsophageal cancer, and as evidence of its rarity says he has been able to find only two other cases in literature, namely, one by Buqnoy, in the 'Bulletins de la Société Anatomique de Paris,' 1855, p. 280; and the other by Lancereaux, in the same 'Bulletins,' 1861, p. 296.

An analysis of the nine cases recorded by Altmann (4), Zahn (2), Buqnoy, Lancereaux, and myself gives the following facts, which are of some interest.

[1] 'Virchow's Archiv,' 1889, Bd. cxvii, p. 221.

The *ages* of six cases were between 50 and 55, two were between 60 and 65, and one was only 42. They were all *males*.

In three of Altmann's cases no history of the illness or manner of death is given. In four of the remaining cases death took place from sudden profuse vomiting of blood, and in two of these at least dysphagia had been the prominent symptom beforehand.

In Buqnoy's case there was dysphagia of four or five months' duration. A bougie was passed as far as, but no farther than, the cardiac orifice; and in the evening there were symptoms of suffocation with vomiting of blood. Two days after this blood was vomited again, the next day the same but less, the next day he had bloody sputa, and died in the night—that is, the fifth night after the passage of the sound. In my own case the symptoms were not those of a simple œsophageal epithelioma; the patient appears to have had epigastric pain, retching, vomiting, cough, and expectoration rather than vomiting of blood. Indeed, there was no history of the discharge of blood from the œsophagus even on the occasion of his death. I have always supposed that he bled into his stomach, and that his stomach was too much distended or too much paralysed by the general collapse to reject it.

Blood was found in the alimentary canal in nearly all the cases. In seven cases there was blood in the stomach, in three of these in the intestines also. The œsophagus is said to have contained blood in two cases, but in one of these (Buqnoy) the stomach is not mentioned as containing it. The quantity of blood-clots or bloody fluid in the stomach is given in four cases: 23 oz. in my own case, 1500 c.c. (about 50 oz.) in one of Altmann's cases, 1000 c.c. (about 33 oz.) in another, and 1800 c.c. (about 60 oz.) in one of Zahn's cases.

In the seven cases in which information is given as to the position of the perforation in the aorta, it was situate in the lower part of the arch or the upper half of the thoracic aorta. In two instances it was near the origin of the subclavian artery. In each of two cases there were two apertures. The condition of the coats of the aorta

around the aperture is also of some interest, as in six cases out of the nine the aperture was in the centre of a patch of diseased aorta, and in some of these cases it is described as endarteritis. Zahn points out that, in his two cases, as well as in Lancereaux's, the coats of the aorta, weakened by endarteritis and thinned on the outside by the invasion of the growth, gave way to the pressure of the blood, and burst from within outwards. In Lancereaux's case the condition seems to have been similar, and the aperture in the aorta was a transverse fissure four fifths of an inch in length.

In only one case (Zahn) besides my own did the cancerous growth project into the aorta. "On the upper wall of the arch of the aorta, near by and internal to the left subclavian artery, is an oval aperture, which measures 7 mm. in a longitudinal and 5 mm. in a transverse direction, and has a somewhat ragged edge. From this projects a mass of tissue somewhat larger than a pea, soft, reddish, and adherent to the left border of the aperture."[1]

A case was shown by Dr. Hale White at the Pathological Society on May 2nd, 1893, in which the aorta was perforated, so as to communicate with a gangrenous cavity in a large sarcoma of the left chest. The œsophagus also opened into the cavity. Death took place from hæmorrhage.

Case 2. *Epithelioma of the bronchus and lung, resulting in a large pulmonary abscess.*—Wm. W—, æt. 43, was admitted under my care, January 29, 1891, for pain in the front of the chest and cough. He has enjoyed good health till the present illness, except for occasional slight colds. Twelve months ago he caught cold, which was preceded by rigor and malaise. There was only slight cough, but abundant expectoration ; no pain in the chest. He was six weeks in bed, and ten weeks before he resumed his occupation. Five months after this he had a similar but slighter attack, lasting two weeks. It was accompanied by slight pain in the chest. Eighteen days ago he was again troubled with cough, being obliged to leave his work, but

[1] Loc. cit., p. 224.

he did not go to bed. There has been more pain in the chest than in the previous attack, the pain not more on one side than the other. He says he has expectorated a small quantity of blood on several occasions during these attacks. There has been no sweating. Though he speaks of these three separate illnesses, he allows that he did not completely recover his health in the intervals.

*Present condition.*—A rather thin, anxious-looking man, tall and dark. He complains of slight pain over the lower part of the front of the chest. He has a cough more troublesome at night than in the daytime; it is relieved when he is sitting or standing. Expectoration is abundant, of a light greenish-brown tinge. The chest is slightly flattened at the upper part of the left side in front. Breath-sounds and resonance are normal over the front; behind resonance is normal on the left side: there is slightly hollow breathing just internal to the lower end of the scapula. On the right side there is dulness below the sixth rib, behind and below the seventh rib in the mid-axillary line. There is loss of tactile vocal fremitus and breath-sounds below the sixth rib behind, and bronchophony at a point above and internal to the spine of the scapula. No ægophony. Precordial dulness is diminished. The pulse is full and soft. Heart sounds normal. Urine normal. Temp. 99·2°.

An exploring needle was introduced into the ninth space behind, and the syringe withdrew some thick offensive pus.

January 31st.—Mr. Jacobson operated to-day by an incision three inches long in the ninth space opposite the angles of the ribs. A pair of dressing forceps was then inserted, and the opening enlarged in all directions. About 10 ounces of foul-smelling pus were evacuated. The cavity was irrigated, a drainage-tube was inserted, and dressings applied. The cavity was felt to be irregular on its interior, and lined by a gelatinous material which was not easily detached. It was about 4 inches in diameter and 2 inches deep. The discharge came through the dressings at night; the outer dressings were changed.

February 3rd.—In front the voice-sounds and breath-sounds are deficient at and below the fifth space; behind there is good entry of air down to a point midway between

the spine and the lower angle of the scapula. Normal voice-sounds with a few râles on inspiration. Below this there is high-pitched note on percussion, with impairment of breath-sounds and loss of voice-sounds.

5th.—Patient is dressed twice a day; the discharge yesterday and to-day was less than on previous occasions, and quite sweet. Appetite fairly good.

17th.—Discharge gradually decreasing; patient seems much brighter than when he entered the hospital. Face not so drawn, tongue clean, appetite good.

19th.—Discharge amounted this morning to about 4 oz., consisting of sero-sanguineous material with but little pus. The exit of the discharge was temporarily blocked yesterday. The temperature, which been mostly between 98° and 99°, rose on the evening of the 17th to 100·2°.

23rd.—Probe inserted into the wound passes forward and slightly inward to the extent of about 3 inches. Laterally it can only move the width of the drainage-tube. Two days later a curved probe could be passed upwards and inwards for about 4 inches.

27th.—Much discharge on the dressings this morning. The greater part was blood, which was still oozing; it ceased on removal of the drainage-tube.

March 1st.—While the patient was about in the afternoon the dressings became soaked with blood. Oozing was stopped by redressing the wound. No fresh hæmorrhage was noticed till nine o'clock the following morning. This was only stopped by withdrawing the tubes and applying pressure.

3rd.—No hæmorrhage since. This morning about 2 oz. of discharge and only a very small quantity of blood.

10th.—Since the 4th the blood in the dressings has varied from 1 to 1½ oz., with about half an ounce of pus.

16th.—Hæmorrhage again last night. The probe passed into the wound does not get beyond an inch and three quarters from the surface. Breath-sounds are heard as far down as the level of the wound. In the evening hæmorrhage amounted to more than a pint, and the wound was plugged.

17th.—About half a pint of blood on the dressings this morning.

18th.—Patient expectorated 2 oz. of bright frothy blood in the night.

19th.—Mr. Jacobson plugged the cavity last night.

The hæmorrhage continues, as well as the expectoration of bright blood. This morning, at ten o'clock, Mr. Jacobson prolonged the original incision backwards, and removed pieces of two ribs above. He thus opened a cavern-like space, from which clot and blood came out freely, the greater part of which was probably recent. When about one pint of this had been removed, a good deal of thicker, darker and more tenacious clot, also more adherent to the walls of the cavity, and of a pale grey colour, was got out. The cavity was then syringed with a hot solution of perchloride of mercury (1 to 2000). There was free hæmorrhage from some recently cut vessels, but the cavity was firmly plugged, and nine Spencer-Wells' forceps were left in position.

The patient was very much collapsed, and died at 1.35 p.m.

The following accounts of the post-mortem inspection and of the microscopical examination of the lung have been kindly supplied me by Mr. J. H. Targett.

*Autopsy.*—The left lung was normal. The right lung was universally adherent, but the adhesions were not very tough except round the wound. After the lung had been removed it was found that the wound communicated with a cavity in the base of the right lung, and that the right pleural sac was entirely obliterated. This cavity in the lung was about four inches in diameter, and had a rugged, sloughy interior. The division of the bronchus which opened into it, was much narrowed by a new growth in its walls. The bronchial glands were swollen and juicy, and one of the mediastinal glands was greatly enlarged by secondary deposits, forming an oval tumour two inches in length by one inch across. It was situated between the trachea behind and the superior vena cava in front, but it was not adherent to the walls of the vein, though it must have caused some compression. The remaining viscera were anæmic but healthy. There was no appearance of growth in any other part of the body.

*Further examination of the specimen.*—After hardening in spirit a longitudinal section of the right lung was made through its root and lower lobe. The inferior division of

the right bronchus was thus bisected. The new growth began opposite the bifurcation of this inferior division of the bronchus, and extended downwards and inwards as far as the cavity in the lower lobe, a distance of 1½ inches. The growth formed a coating half an inch thick round this portion of the bronchus, the calibre of which was considerably narrowed where it opened into the pulmonary cavity. This cavity occupied a large part of the lower lobe of the lung. It measured after immersion in spirit 3 inches from above downwards, 3½ inches from before backwards, and 2½ inches from side to side. Externally it was bounded by thickened and adherent pleura, and a thin layer of pulmonary tissue; inferiorly by a thin layer of lung tissue and adherent diaphragm. The aperture in its external wall resulting from the first operation measured 2 inches by 1½ inches. The interior of the cavity was nodular, and crossed by bands of lung tissue at the upper end. It contained a small amount of blood-clot and sloughing material, to which the hæmorrhage was apparently due. A large branch of the pulmonary vein running with the affected bronchus was quite obstructed by new growth. The remainder of the lung was healthy.

The portions of the lung submitted to histological examination were—

A. *The wall of the affected bronchus.*—The section was composed of dense fibrous tissue permeated by branching processes of epithelial cells. These processes were thus cut in various directions, giving rise to areas of different sizes and shapes, some of which communicated with one another. The cells within these areas were for the most part large and flat like squamous epithelium, and their outlines were angular from mutual pressure. At the periphery of these spaces the cells presented none of the characters of cylindrical epithelium. Much of the growth had undergone degenerative changes, and the cells in consequence looked blurred and stained imperfectly. A large pulmonary vessel (probably an artery) was included in the section. Its walls were compressed, and its calibre was occupied by an organised thrombus. The growth surrounded the vessel on all sides,

and in some places extended halfway through its middle coat.

B. *The wall of the pulmonary cavity towards the diaphragm.*—This section consisted of a thin layer of lung tissue and the muscular substance of the diaphragm, to which it was firmly adherent. The former was almost entirely replaced by growth, in which groups of dark pigment granules were met with—the remnants of the original pulmonary tissue. The character of the growth itself was similar to that in the previous specimen, but the long branching epithelial processes were better marked, and among the squamous cells were many small well-defined "bird's-nests." The layer of growth nearest to the pulmonary cavity was caseous and badly stained. The diaphragm was unaffected.

C. *A nodule projecting from the upper wall of the cavity.*—This showed traces of the original lung tissue, pigment granules and nodules of hyaline cartilage of the bronchioles. But the greater part of the section was composed of new growth, closely resembling that described above. Here the squamous character of the epithelium was remarkably distinct, and quite beyond all doubt, while the "bird's-nest" formations were large and numerous. The pieces of cartilage embedded in the growths were becoming invaded at their margins.

From the character of the cells, their mode of growth and extension, and the presence of "bird's-nests," we may venture to conclude that the growth in the bronchus and the walls of the pulmonary cavity is a squamous-celled epithelioma.

*Remarks.*—The points of interest about this case seems to me to be the nature of the growth, the suppurative change which took place in it, the resemblance which it bore clinically to an empyema, and the fatal termination by hæmorrhage. As is well known, primary cancer of the bronchus or lung is not by any means common, and in this case the epithelial malignant growth probably began in the bronchial mucous membrane, and spread thence to the lung tissue, while one of the mediastinal glands was enlarged by secondary deposits.

I have not thought it necessary to search widely for other

cases of primary cancer of the lung. Reference to some will be found in Ziegler's 'Text-book of Pathology.' I may, however, refer to the special origin of the pulmonary cancer in disease of the bronchus, which is illustrated by the case. An instance of this was brought before the Pathological Society in 1889[1] by Dr. Henry Handford, of Nottingham, and I had the privilege of reporting upon it as a member of the Morbid Growths Committee, in conjunction with Dr. Charlewood Turner. The patient presented indications of obstruction of the left bronchus, which was attributed to a new growth or to aneurism, and ultimately profuse hæmoptysis occurred, of which he died on the fifth day. The mucous membrane of the trachea was affected by carcinoma about three quarters of an inch above the bifurcation; and the growth had spread along the left bronchus, nearly filling up its lumen, but without affecting the outside at all. The upper lobe was collapsed, and contained nodules of new growth; the lower lobe only showed one or two white fibrous-looking bands spreading along the bronchi, which in some instances were completely obliterated. There were nodules of new growth on the left visceral pleura, and in the liver. The mediastinal and bronchial glands were enlarged. The growth was typically carcinomatous. Dr. Handford refers to some other cases of primary cancer of the bronchi by Biermer, Rokitansky, and Langhans.

My case differs from Dr. Handford's in the nature of the growth, a squamous epithelioma, and in the invasion of the lung, being by means of an apparently large infiltration, rather than by small nodules. The calibre of the bronchi was "considerably narrowed;" to what extent it produced collapse of the lung, or what was the condition immediately preceding the abscess formation, may be doubtful.

From the clinical, as much as from the pathological point of view, this suppuration must be regarded as of great interest. The poor vitality, so to say, of cancerous growths is familiar to us, and there is no need to remind ourselves of the facility with which they caseate, soften, or break-down when in the substance of organs, or ulcerate and become detached in fragments when they form on mucous

[1] 'Path. Trans.,' 1889, vol. xl, p. 40.

surfaces, such as those of the rectum and œsophagus. But the formation within the extent and circumference of the growth of a definite collection of pus, indistinguishable to the naked eye, at least from the pus of advancing acute abscesses, is by no means so frequent a phenomenon. Suppuration is probably more common in the abdomen than elsewhere in connection with cancers of the bowel, and I remember a surgical friend telling me of his having to do with three cases of suppurating intestinal cancer within a very short period. In all probability local conditions favouring sepsis must be looked for to explain its occurrence. Such antecedents can readily be conceived in the case of intestinal cancer, and it occurs to me that here also similiar unfavourable circumstances may have arisen, in the fact of the affecting lung being connected with an obstructed bronchus. One of the results of the obstruction of a bronchus is, as one well knows, the dilatation of the distal portion of the tube, so-called bronchiectasis ; and dilated bronchi are constantly the source of a purulent fluid, which is not infrequently highly offensive and septic.

Turning now to the clinical aspects of the case, these were no doubt very much determined by the suppuration, which occurrence we have been discussing. The difficulty of distinguishing malignant disease of the lung from pleuritic effusion has been frequently experienced ; so much so that it is recognised that in elderly patients the possibility of malignant disease should be always borne in mind in the case of a disease of some standing which resembles pleuritic effusion ; and this, not only because a pleural effusion is often due to malignant disease, and thus the two coexist, but because, by the conversion of a portion of the lung into cancer, the physical signs and conditions of pleural liquid may be almost exactly simulated. But here the resemblance to pleural liquid was assumed by a mixed condition of solid and liquid in the lung itself. In the former case an exploration with the needle may suffice to show that there is no pleural fluid, and that there is probably malignant disease ; in the present instance the syringe withdrew some thick pus, and thereby perpetuated the view that we had to do with an empyema. There appeared to be no room for doubt ; the patient was

very ill; he had the physical signs of empyema; he had a history which, so far as we knew its details, was compatible with it, and we proceeded to deal with it in the usual way by incision. The removal of the pus seemed to be imperative.

Practically, so far as physical signs are concerned, an abscess can scarcely be suspected till it is realised that there is a cavity. Dr. Wilson Fox says,[1] "The signs of an abscess are necessary to the diagnosis of abscess; but they are not always markedly apparent nor present in all cases to the same degree." And these signs are of course the familar amphoric breathing, pectoriloquy, and gurgling râle, which we know so well in the larger tubercular cavities. Such signs were absent in my case; the nearest approach to them was "slightly hollow breathing, just internal to the lower end of the scapula," but as bronchial breathing is not unknown in pleuritic effusion, this did not awaken any special attention.

It is interesting to ask how the cavity could have been maintained so full of pus when the patient was expectorating a good quantity, which must have come from this source. It is possible that the narrowing of the lumen of the bronchus by the growth may have helped to prevent more than the excess of secretion from being evacuated.

The last point for our consideration is the mode of death, which was by hæmorrhage. This is of interest in connection with Dr. Handford's case, already mentioned, and with two other cases of carcinoma of the lung shown by him before the Pathological Society in the preceding year.[2]

In all three death was the result of profuse hæmorrhage, and Dr. Handford mentions them as exceptions to the rule which has been formulated that new growths involving the trachea and bronchi do not commonly destroy life suddenly and unexpectedly by hæmorrhage. In this last respect my case must be allowed to differ a little from those of Dr. Handford, though the point is perhaps not a very essential one. The bleeding, though ultimately profuse, was only slight at first, so that the patient did not die till the twentieth day after the first appearance of unmixed blood. What

[1] 'Treatise on Diseases of the Lungs and Pleura,' 1891, p. 409.

[2] 'Trans. Path. Soc.,' 1888, vol. xxxix, p. 48.

share the surgical manipulations and dressings may have had in the hæmorrhage must be doubtful, but probably, in any case, it would have eventually taken place. In one of Dr. Handford's cases the patient brought up 20 or 30 ounces on each of three consecutive days, and died thirty-six hours after the last hæmorrhage. In another of his cases death took place in about twenty-four hours from bleeding, and the third patient had "a copious hæmoptysis, which proved speedily fatal."

In the last volume of these 'Reports,' in the list of specimens added to the pathological museum of Guy's Hospital during the year 1891, is the description of one, presented by Dr. Newton Pitt (No. 3349A), which is in some respects like mine. It is a lung excavated as the result of the growth of a squamous epithelioma; the patient died from exhaustion after many months' illness. The account is as follows:

*Epithelioma of the lung with cavitation.*—A left lung, the lower two thirds of which is hollowed out, forming a cavity measuring 4½ inches in its vertical and antero-posterior diameters. The thin layer of pulmonary tissue and pleura which forms the wall on its lateral aspect has been partially removed, and shows the opening of the main bronchus into the cavity, which is indicated by a blue rod. In the recent state the interior was shaggy, and covered with muco-purulent secretion. On the reverse of the specimen, a section through the consolidated base of the lung displays sinuous cavities resulting from dilatation of bronchial tubes. The pleura over the lower lobe is much thickened and was adherent. Histological examination of the wall of the cavity shows the pulmonary tissue to be infiltrated by a malignant growth having the characters of a squamous epithelioma.

William A—, æt. 68, was an out-patient for some months, suffering from profuse, blood-stained, muco-purulent expectoration, the quantity of secretion sometimes exceeding a pint in the twenty-four hours. This condition continued for twenty months, towards the end of which time the patient became affected with anasarca. He died from cardiac failure. The post-mortem examination was incomplete.

Case 3. *Multiple cancer of the spine and ribs secondary to scirrhous cancer of the breast.*—Edith W—, æt. 54, admitted February 12, 1889, for progressive weakness and wasting, enlargement of glands on both sides of the neck, and sharp shooting pains in the left shoulder, running down the arm to the tips of the fingers.

*Personal medical history.*—Had scarlet fever, measles, and whooping-cough when a child; always been more or less subject to bronchitis since the scarlet fever, until the last three years. About nine weeks ago, after doing a day's washing, had pains of an "excruciating" character come on between the shoulders; it was "like daggers going through her;" she never noticed anything of the kind before.

A week or two after (about two months ago) the pain seemed to be getting into the right shoulder, and shortly afterwards she noticed there were two or three little lumps on the right side of the neck. They were tender to the touch, but she cannot say whether or not the skin over them was red.

About three weeks ago pain in the right shoulder ceased, but was felt in the left, where it has remained ever since, occasionally shooting down to the finger tips. She noticed small lumps on this side, which were also tender when touched; but she did not discover them till after the pain came on in the left shoulder. She cannot say whether the lumps have increased in size or number since she first discovered them, but she states that she has been progressively getting weaker and losing flesh since the first appearance of the pains.

During the last week or two the bronchitis has appeared again. She has never spat up blood; she has no cough now, and does not constantly expectorate. The sputa, when present, are "clear and frothy."

*On admission* the patient looks rather thin, and her complexion has a somewhat sallow tint. She has dark hair turning grey, grey eyes, no arcus senilis. The pupils are normal. The fingers are not clubbed; there are a few dilated venules on the cheeks. The skin hangs loose in some parts, as on the abdomen, neck, and face. In the posterior triangle of the neck on both sides there is a group of enlarged super-

ficial cervical glands, five in number on the left side, and in one mass on the right. They have not coalesced with one another on the left side, and are firm but tender to the touch; there is no redness of the skin over them. No enlarged glands anywhere else can be made out. Thyroid body not enlarged.

The tongue has a brownish-white fur in the centre, and is red at the tip and edges. Deglutition normal. Appetite bad. A feeling of nausea "at times." No particular thirst. Bowels constipated. No hæmorrhoids. No ascites. The liver dulness commences in the fifth space, and becomes absolute over the seventh rib, dulness extending below the margin of the ribs, where the edge of the liver can be felt.

Both sides of the chest move equally and well. Tactile vocal fremitus seems slightly diminished at the left base, and the patient flinches and complains of tenderness on percussion there. Percussion note normal. Breath-sounds somewhat harsh. Vocal resonance is not so distinct at the left base, but is slightly increased at the right apex. A few rhonchi can be heard occasionally; no râles; no cough or expectoration. Respirations 28 per minute.

Heart sounds distinct. No bruit. Impulse felt in the fifth space in the nipple line. Precordial dulness slightly increased to right and left. Pulse 132, irregular.

The spleen cannot be felt.

She cannot turn her head from side to side, and the head tends to fall forward when she sits up. She cannot hold anything in her left hand. She says she cannot stand without support, as she has become so weak, and, indeed, she was carried into the ward. The left leg seems to her the weaker. She can move her legs about in the bed and there is no anæsthesia or analgesia. She can exert a fair amount of power against resistance, the right leg being the stronger. Plantar reflexes and knee-jerks are present, the latter exaggerated. There is no very marked wasting of the trapezii. The muscles of the legs are not much wasted, but those of the upper extremity are more so, especially the deltoids. Pains shoot down the left arm from the shoulder to the finger tips. The pupils react to light and accommodation. Smell and taste are unimpaired. Hearing is slightly defective.

February 12th.—Ordered Pot. Iod. gr. v, Mist. Acid. Co. ad ℥j, t. d. s.

14th.—Salicylate of sodium was added to the mixture, and glycerine and extract of belladonna were applied locally.

19th.—The pains were relieved, but she was fully under the influence of the salicylate, having delirium with hallucination, and on the subsidence of those symptoms the pains returned.

22nd.—On testing her power of standing it was found that she had to be supported, but could stand with only slight assistance, and she could move her legs pretty well, but walking caused her great pain between the shoulders. She held her head slightly forward, and her shoulders raised. There is no pain in the lower extremities, but pain on pressure over the spine ; a good deal of pain between the spines of the scapulæ, in the abdomen and in the arms.

27th.—Sharp pains at frequent intervals in the right hypochondriac and lumbar regions. She lies in bed with her thighs flexed on the abdomen. Her breathing is mostly thoracic, and she looks anxious. The abdominal walls are rigid, and nothing can be felt.

March 2nd.—The chest is tender to percussion in front, and there is some dulness near the right axilla, where the tenderness is greatest. Slight vocal fremitus. There do not appear to be any isolated spots where there is marked dulness. At the apices the breathing is rather harsh, but no bronchophony. At the back, good resonance over the right lung. Over the left lung there is some dulness, especially marked at the base and between the spine and scapula. The breathing over these regions is somewhat bronchial in character. Rhonchi heard in many places. No evidence of effusion or pleural adhesions. Great pain on coughing over the region of the left scapula. Abdominal pains diminished.

6th.—*Dynamometer :* right hand 20, left hand 10. There is still pain over the left scapula running down the left arm, but there is none down the right arm. She has wasted considerably since admission, and the deltoids and muscles of the arm are atrophied, but not the trapezii. The cervical portion of the spine is thickened, especially at the lower end, and the head is drawn forward, so that she is unable to straighten the neck or draw the head backwards.

During the rest of the month of March she suffered from the dull aching pains extending from the shoulders to the wrists, and from a feeling of constriction round the lower part of the waist. The lower end of the sternum and some of the ribs now became tender, and a swelling was observed on the left seventh rib at the seat of tenderness. An examination of the right breast revealed the presence of an indurated mass in its substance about the size of a walnut, over which the nipple was retracted. Dyspnœa now became a prominent symptom, but the only physical signs were slight dulness on percussion, with deficient breath-sounds at the right base and a few râles at the left base.

From this time the patient suffered severely, partly from dyspnœa, partly from the pains down the arms, in the sternum, and round the waist. She lay either on her side or semi-recumbent, but always with the head bent forward upon the chest, and with the legs flexed towards the abdomen. She was unable to raise the head or to bend it from side to side. She had very little sleep and lost flesh rapidly. The physical signs of impairment of the lung were not very pronounced, and consisted chiefly of slight dulness and bad entry of air at the right base, with scattered rhonchi. The respirations varied from 30 to 38, the pulse from 140 to 160.

In the latter part of April the left arm became numb, more powerless, and œdematous. The sternum assumed a peculiar shape, due apparently to the pressure of the chin upon it ; that is, the upper part was driven back and became concave while the middle part was bulged forward and convex. On the ribs have recently developed a number of nodules, obviously due to malignant growth. These are situate posteriorly, near the angles of the ribs. On the right side one is felt on the seventh rib, one on the eighth, one on the ninth, one on the tenth, and two on the eleventh. On the left side there are three on the ninth rib, one on the tenth, and one on the eleventh rib, thus making eleven in all. These nodules or thickenings of the ribs are about one inch in diameter, and form visible prominences on the wasted chest.

On May 3rd the face, especially the upper eyelids, became

œdematous. With increasing dyspnœa and cyanosis, she died on May 5th.

The post-mortem was made on May 7th by Dr. Newton Pitt. There was a marked flexion forwards of the neck, with absence of the usual curves. This on section was found to be due to growth infiltrating the seventh and eighth cervical, and the first, second, and third dorsal vertebræ. The masses of growth projected as three diffuse rounded masses into the spinal canal, where they compressed the cord slightly; they corresponded to growth adjacent to intervertebral discs, but in the bone. An antero-posterior section, through the spine, showed that one of the vertebræ, which was infiltrated with growth, was softened and much reduced in thickness. In the centre it was reduced to one eighth of an inch in thickness, while posteriorly, where the growth projected into the spinal canal, the thickness was a quarter of an inch.

The dura mater was unaffected, except that the uppermost part was thickened to one twentieth of an inch. There was a mass of flattened growth one and a quarter inches in diameter attached to the falx cerebri, anterior to the quadrate lobule; it adhered to the pia mater on either side, but had not infiltrated the cortex. The brain was wasted, but otherwise normal, and weighed 43 oz.

The condition of the ribs was the most striking feature of the case. Large masses of calcified growth occupied the central portion of the ribs and distended them. Some of these growths were one inch in diameter at the centre, and two to three inches long, and were dense calcareous masses. The rest of the ribs near them, however, had undergone a remarkable atrophic change. They were very flexible and fragile, and readily broke; and they were much thinner. On the right side were one or more fractures of ribs from the second to the ninth inclusive; on the left side the fifth, tenth, and eleventh. It is possible that some of these fractures took place during the examination. Nowhere had a fracture occurred through one of the masses of growth.

The masses were most palpable on the inner surface. They had invaded the ribs from the fourth to the eleventh

on the right side, and from the sixth to the eleventh on the left side.

The sternum was readily flexible in the middle, and was found to be infiltrated with soft masses of growth which were not calcified, and which somewhat enlarged the antero-posterior diameter.

*Lungs.*—The upper lobes were emphysematous and distended, especially the right. On the right lung were numerous dense white nodules of growth, which occurred chiefly between the middle lobe and those above and below, and had spread on the surface by contiguity. The pericardium was also invaded by small masses.

Left internal jugular vein contained ante-mortem thrombus.

The cervical glands contained growths, and nodules of growth in the anterior mediastinal glands had compressed the innominate vein, in which, however, there was no growth.

The liver contained innumerable secondary growths, mostly dense white spherical nodules the size of marbles, which shelled out easily with the finger ; they infiltrated both lobes. Some glands in the portal fissure infiltrated with growth were in contact with the bile-duct, but did not compress it. Growth in aortic and lumbar glands surrounding aorta. No thrombosis of vessels.

Kidneys, 9½ oz. The right breast was small, atrophied, and infiltrated with a growth which microscopically was a scirrhous carcinoma. Left breast normal. No growths in the limbs.

*Remarks.*—The above case tells its own tale. A primary scirrhous carcinoma of the breast was followed by deposits in the cervical vertebræ, the ribs, sternum, lung, and falx cerebri. The most striking features of the case clinically were, first, the curious deformity which resulted from the disease of the spine, the forward position of the head, and the yielding of the sternum to the pressure of the chin. For the last few weeks of her life the patient was in the fixed position, and the depression of the upper part of the sternum was obviously due to a softening of its structure which allowed it to yield, while the central projection was prominent by contrast. The clinical symptoms pointed early to compression of the roots of the spinal nerves by the falling

together of the softened vertebræ upon them as they passed through the intervertebral foramina. The examination showed that the change in the vertebræ was not only one of softening, but also of absorption, since one vertebra was represented only by a very thin layer (one eighth to a quarter of an inch) of new growth between the intervertebral discs; but while it was thus unable to spread upwards or downwards on account of the resistance of the intervertebral discs, the growth projected in three nodules, corresponding each to the body of a vertebra, into the vertebral canal. The nervous system must have been injured in two ways: (1) by the compression of the cord, which Dr. Pitt observed, however, was slight; and (2) by the compression of the nerve-roots, which would be expected as the vertebræ fell in upon one another, and which was evidenced during life by the pain down the upper extremities from the shoulder to the finger tips.

Case 4. *Cancer of the right kidney and spine; small (? atrophied) pancreas; deficiency of colouring matter in the fæces.*—Martin Y—, æt. 62, was admitted under me on July 16th, 1890, for pain in the epigastrium, loss of appetite, and wasting. He has been a labourer working in a timber yard for thirty years. He has not been in the habit of drinking. There is nothing in the family history bearing on the case. He has had measles and smallpox, and gonorrhœa at the age of twenty-four.

Two years ago the patient first began to feel a pain in the lower part of the chest and epigastrium, which became worse after meals. It was not at first very severe, and he did not pay much attention to it, but it gradually got worse. It was usually of dull, aching character, but it sometimes became sharp and shooting, and stopped the patient's breathing. In August, 1889, he became suddenly worse, and had to take to his bed. He was attended by doctors without relief, and he has now been in bed since February last with continued pain in the epigastrium and complete want of appetite, but without sickness or flatulence. He has lost a good deal of flesh. The bowels have been constipated, sometimes not open for eight or nine days. For eighteen

months he has had pains shooting down from the buttocks to the feet. In 1886 he passed blood in his motions for six months.

The patient is pale and emaciated, with a careworn expression. The face, forearms, and hands are covered with scars and scabs produced by the splashing of creasote on those exposed parts during his work. Some enlarged glands can be felt in the axillæ and groins.

He has a very poor appetite. The tongue has a yellow fur on the dorsum, and red edges; the breath is fœtid, and the bowels are constipated. He has pain of a dull aching character in the epigastrium, increased by pressure. The abdomen is retracted and the skin flaccid. The pulsation of the abdominal aorta can be felt readily, but there is no tumour or anything abnormal in the epigastric region. The left side of the abdomen is normally resonant; on the right side hepatic dulness extends below the ribs nearly to the level of the umbilicus. Splenic dulness is normal.

The chest is hyperresonant on both sides in front, and the entry of air is somewhat deficient at the left base. He has occasionally expectorated a little blood.

There is no precordial dulness, and the heart sounds and impulse are masked by the lung. Pulse 60, regular, full. Urine clear, neutral, sp. gr. 1010; no albumen, blood, or sugar.

He was ordered an enema on July 18th, passed a large quantity of dirty white, clay-like, soft masses and mucus. He was ordered Tinct. Opii ♏v, Sp. Ammon. Arom. ♏xx, Aq. ad ℥j, t. d. s.

July 24th.—He has continued to have pain in the epigastrium, and pain and tenderness over the right side of the chest below the nipple. To-day he passed a soft colourless motion.

29th.—He complains of a shooting pain in the buttocks running down to the foot. This morning he passed a loose motion similar to the previous one—that is, white or colourless.

August 3rd.—His epigastric pain is less, but he has pain on pressure in the right lumbar region; nothing, however, can be felt there, and the only notable feature about the

abdomen is the rigidity of the abdominal muscles, which remained throughout the case. To-day he passed a quantity of thick liquid granular stuff of a dirty light brown colour with some harder masses, but no obviously undigested food. The residue, obtained by shaking up some of the fæces with ether, pouring off the supernatant fluid, and evaporating, stains black with osmic acid.

During the rest of the month of August his symptoms were sleeplessness, epigastric pain, pains down the thighs, and towards the end diarrhœa and sickness. The colour of the motions was dirty light brown, or at most pale yellow, and they were liquid and offensive. The diarrhœa was checked by a logwood, chalk, and opium mixture.

On September 14th he passed a motion consisting of white pasty-looking masses, and on the 15th an abundant cream-coloured motion of the consistence of thick gruel.

18th.—I examined him carefully, and found the spines of the vertebræ rather prominent; tenderness on pressure at a small spot the size of a penny at the outer border of the erector spinæ, and 1½ inches above the iliac crest; also tenderness over the right sacro-iliac joint. The percussion note at the right base was not quite so good as at the left. The pain in the leg starts from the lumbar region on the right side, runs down the front of the thigh and the inner side of the leg to the ankle.

He continued to pass white motions, to vomit occasionally, and to have "sciatica" during September and October.

On November 10th he had lost 7 lbs., weighing then 6 stone 12 lbs. The stools on November 25th were slate-coloured, somewhat relaxed; on November 27th formed, of faint yellow colour. On December 2nd the fæces were formed into thin coils; they were divided longitudinally into two portions, one being of faint yellow colour, the other of slate colour and not quite so solid.

A careful examination on December 18th revealed only some tenderness over the last rib, and deficient entry of air at both bases.

On December 31st the urine was for the first time abnormal. It was acid, of sp. gr. 1010; contained albumen, blood, and casts.

January 5th.—Still complains of pain down the leg. Motions loose, of light clay colour. Face slightly yellow and dark under the eyes.

9th.—For some time now his cough has been troublesome, and he expectorates in the day about 1½ oz. of frothy pale green mucus.

12th.—Urine acid, sp. gr. 1015, very small amount of blood and of albumen. Motions much darker, greenish grey.

He gradually sank, and died on January 18th, 1891. Except for a temporary rise during the first few weeks of August, his temperature was mostly within normal limits.

*Autopsy.*—On opening the abdomen there was found a mass of secondary growth, thus described by Mr. Targett :— To the right of the lumbar spine and in the psoas muscle, was a large secondary deposit, which had invaded the bodies of all the lumbar vertebræ save the first. Those of the third and fourth were most affected, and between their transverse processes the growth had extended backwards into the erector spinæ. After the spinal canal had been opened a small mass of growth was found on the back of the bodies of the third and fourth lumbar vertebræ, but it was quite free of the dura mater, and had not obviously compressed the cauda equina. The vena cava and right common iliac veins were displaced forward by the growth, but not otherwise affected.

The intestines were very thin, and the omentum almost destitute of fat. In the lymphatics was chyle, as one sees when the process of digestion is going on. The intestines contained a moderate amount of material, and were healthy. The mesenteric glands were greatly enlarged by the growth, and around the head of the pancreas were several the size of pigeons' eggs. They were very hard; it seems possible at first that they had pressed upon the pancreatic duct, but further investigation showed that the duct was about the ordinary size, just admitting a small probe, and nowhere dilated or stenosed. The pancreas was, however, small, not more than half its usual size; and the cause of this is not clear, unless it was diminished in proportion to the general emaciation. There was no trace of growth in it, and though atrophied it seemed normal in structure.

The liver was of normal size, and contained two very insignificant nodules of growth, each about the size of a pea, one on the under and another on the upper surface of the right lobe. They were depressed in the centre. The gall-bladder was very much distended with bile, and was about 3½ inches long. The bile was black and viscid, but could be squeezed out of the papilla without much difficulty. The ducts were normal.

Spleen 4 oz., firm ; no secondary deposits.

The left kidney weighed 4 oz., and was pale but normal. The right kidney was much larger, about two thirds of it consisting of a growth the size of a cricket ball, which occupied the middle part of the organ. The cut surface of the growth was very firm, showed extensive caseation or necrosis, and here and there hæmorrhages. The cellular tissue in the hilus of the organ was œdematous. The capsule peeled readily from the surface of the organ. The right pleura was inflamed, and contained 12 oz. of fluid ; the lung was compressed, and showed some pus in its tubes.

*Microscopical examination.*—The kidney showed the appearances of an ordinary scirrhous carcinoma with spheroidal cells. In parts the growth was necrotic.

In the axillary and mesenteric glands there were deposits of cancer, showing more epithelial cells and a less amount of stroma.

*Remarks.*—There was again in this case the familiar association of pain, deep-seated or radiating along nerve-trunks, with emaciation and prostration in an elderly person caused by cancer ; and so far as the nature of the disease was concerned there was never any doubt. The indications as to the seat of the lesion were, however, somewhat misleading. For my own part, I believed that there was very probably cancer of the pancreas, with secondary invasion of the spine. And I based this diagnosis with some diffidence, I allow, upon the fact that the tumour was not of sufficient size to be easily perceptible to touch, and upon the absence of colour in the motions, while the skin was free from jaundice. The inability to feel a tumour cannot of course be an argument in favour of its presence, but I thought that a tumour in the kidney would certainly have been felt at some time of the

long period the patient was under our observation, while from the position of the pancreas a mass of cancer in its "head" might easily be overlooked. As a fact the examination was always hampered by the constantly rigid condition of the recti and abdominal muscles, and our position would, I dare say, have been more clear if we had early in the case submitted the patient to chloroform, and thus more thoroughly on relaxation of his muscles examined the upper part of the abdomen. I must confess, however, I was influenced most in my view of the case by the interesting cases and remarks of Dr. Walker, of Peterborough, in the paper read before the Royal Medical and Chirurgical Society[1] for 1889, vol. lxxii. He there expresses the belief that the colourless stools, which are generally regarded as being due to absence of bile, and occur in cases of obstructive jaundice, may occur also as a result of obstruction of the pancreatic duct, when the formation and secretion of bile into the alimentary canal are perfectly normal. One of the cases he records in support of this view is especially striking. A gentleman, who died eventually at the age of ninety, passed colourless motions for the last twenty-four years of his life. At the age of eighty he had an attack of pneumonia, during which for a few days he had distinct jaundice, with deep staining of the conjunctivæ, skin, and urine. From these he recovered, and with this exception he never suffered from icterus during his illness. These facts show that his liver was active and could make bile, but that there was no retention of that fluid. At the post-mortem the bile-ducts were patent, but the pancreatic duct was dilated, and one inch from the duodenum it was obstructed by an irregular stone, measuring eight lines long by three or four wide at its widest part.

Dr. Walker's explanation is to the effect that stercobilin, the colouring matter of the fæces, is indeed derived from bile, and that therefore the secretion of bile is necessary to the normal colour of the fæces ; but that the conversion of bile into stercobilin only takes place in the presence of the pan-

[1] 'Medico-Chirurg. Trans.,' 1889, vol. lxxii, p. 257: "On the Clinical Significance of Colourless or Clay-coloured Stools unaccompanied by Jaundice, and their Connection with Disease of the Pancreas; and on the Part played by the Pancreas in Eliminating Bile from the Intestines."

creatic secretion, and that in the absence of the latter the bile-pigment is all absorbed from the intestine, and no stercobilin is formed.

Here it seemed to me was a case in point. My patient was almost certainly suffering from cancer; the pain was in part epigastric, in part referable to the spine and the nerves radiating thence. It was easy to conceive that the cancer spread from the pancreas and involved the spine; it was also not difficult to believe that the tumour might escape detection from its deep position behind the stomach, protected by the abdominal parietes stretching from the costal margin, and by the rigidity of the muscles. The stools throughout were remarkably deficient in normal pigment. They were described on different occasions as white, clay-like, whitish-brown, light brown, slate-coloured, and sometimes faint yellow. Only just before his death were they described as "much darker, greenish-grey."

On the other hand, there was a remarkable absence of the two most characteristic of the features of renal cancer, namely, a tumour in the loin and blood in the urine. I examined him frequently, and never noticed any such resistance in the right lumbar region as to make one suspect renal cancer, though the situation of the pain in nerves descending to the leg and thigh would have been more easily explained in association with such a tumour than with one of the pancreas. But the persistently normal condition of the urine is very striking. It was on December 31st that the urine first contained a little blood and albumen. This was only eighteen days before his death; and, on the other hand, it was two years after the first occurrence of pain down the legs, and two and a half years after the first epigastric pain.

The explanation of the white motions still remains doubtful. The pancreas was neither the seat of cancer, nor was its duct obstructed. It was smaller than normal, but Dr. Perry, who made the post-mortem, did not consider that its structure was in any other respect abnormal. It is to be regretted that a microscopic examination was not made of it, but in the absence of that we must, I think, assume that the succus pancreaticus was poured into the duodenum, and the case,

therefore, does not lend very powerful support to the view put forward by Dr. Walker. On the other hand, the liver appears to have been also normal, except that it contained two small nodules of carcinoma ; and its power to discharge bile into the alimentary canal was shown by the presence of dark-coloured bile in the gall-bladder, which in spite of its viscidity could be forced into the duodenum in the usual way.

Case 5. *Sarcoma of abdominal lymphatic glands, ureter, bladder, duodenum, and head of the pancreas ; hæmaturia ; suppurative parotitis ; death.*—George C—, æt. 45, was admitted under my care on September 12th, 1891. He has been an accountant recently, and before that he was secretary to a company.

In January, 1890, he was treated for piles, and shortly after this had an attack of "rheumatic gout" affecting some joints in both lower extremities.

About four months ago he began to pass blood-clots in his urine ; but for eight or nine months he has had pain in both loins, groins, and thighs, and he has been losing flesh for twelve months. For six months he has been liable to sickness. There is no history of injury to the back.

On admission he is sallow and anæmic, with hair and beard turning grey. His skin is dry, harsh, and burning. He is emaciated ; he readily faints, and has nausea on slight exertion.

The abdomen is rather rigid on the right side. There is fulness in the right loin and right hypochondrium, and there is a circular prominence above and to the right of the umbilicus. On manual examination a tumour is found extending from the right costal margin into the right iliac region, and from the middle line outwards into the right flank. The visible prominence mentioned is a hard circular boss on the surface of the tumour. Below the level of the umbilicus the tumour is situated more posteriorly, and one has to dip deeply to explore its limits. Just below the umbilicus a very distinct, small, hard nodule can be felt. The left margin of the tumour is fairly well defined, lies

just to the left of the middle line, and extends from the epigastrium to the umbilicus, then falls away to the right, becoming less marked. The tumour is not very prominent in the right loin. Bimanual examination shows the continuity of the mass from back to front; it is rather firmly fixed, and it is scarcely moved either by the hands or on inspiration. The surface is mostly dull, but there is a band of resonance presumably due to the colon just below the margin of the ribs, and on a deep inspiration the colon can be seen to descend over, and raise the abdominal wall from off the almost fixed tumour behind it. The patient was unaware of the existence of a tumour in his abdomen.

The urine is of dark brown colour, depositing grumous material, acid, of sp. gr. 1012, containing blood and albumen.

September 15th.—Passed a very bad night; troubled with hypogastric pain and frequent desire to micturate, which mostly results in the passage of a few drops of urine only. During the night he passed three or four clots of blood.

The urine is smoky, of reddish brown sp. gr. 1014, acid, with blood and albumen, and a deposit of altered blood. Under the microscope blood-corpuscles, débris, blood-casts, spindle- and round-cells in abundance, small clots of blood.

He has a sleeping draught of chloral and potassium bromide at night.

18th.—Urine only slightly smoky.

19th.—He has passed four or five large flat decolorised clots; one measured 1 inch by $\frac{3}{4}$ of an inch; another $\frac{1}{2}$ inch by $\frac{1}{2}$ inch. These clots caused intense pain as they passed the urethra.

September 21st.—Several large irregular clots passed *per urethram* with severe pain, some flat, others cylindrical. One of the clots weighed 77 grains, another 71 grains; the latter 4 inches in length, $\frac{3}{4}$ of an inch at its broadest part, and $\frac{1}{2}$ inch in thickness. The flat clots have one edge rounded and regular, and the other irregular and fringed.

23rd.—Two small clots passed; otherwise only the faintest trace of blood in the urine.

24th.—Urine gives no reaction for blood, only the faintest ring of albumen with nitric acid.

25th.—Urine again contains bright red blood.

26th.—Another clot, 3 inches long.

28th.—I examined the rectum, but was unable to feel anything wrong in the direction of the bladder. He did not pass much blood after this. The last clots noted were passed on October 2nd. He emaciated rapidly.

October 12th.—Swelling of right parotid and submaxillary glands. Increasing prostration.

17th.—Death.

The following is Mr. Targett's account of the autopsy:

Body extremely emaciated. Some œdema of the right leg. Head not examined. Right parotid gland much enlarged by diffuse suppuration. Right submaxillary lymphatic glands also suppurating.

*Thyroid* normal.

*Lungs.*—Old scars at left apex with calcareous nodules. Pus in the tubes of the right upper lobe. Both lower lobes very œdematous.

*Larynx.*—Œdema of right arytæno-epiglottidean fold, not enough to obstruct the glottis.

*Heart,* 13 oz. The aortic cusp of the mitral valve was distinctly thickened at the margin, but there was no stenosis.

*Abdominal viscera.*—The central region of the abdominal cavity was occupied by a soft white growth, which was attached to the upper end of the right kidney, and filled the right loin; thence it extended across the spine behind the duodenum and pancreas to the left psoas muscle. The whole mass of growth measured eight inches in length and six inches across. The masses in the right loin reached as far down as the right iliac crest, and these were enlarged glands along the iliac vessels. The pelvis of the right kidney was occluded at its junction with the ureter, and from this spot to the brim of the pelvis the right ureter was embedded in the growth and could not be traced. It again became recognisable as it crossed the pelvis to the bladder, where it formed a solid white cord, in size and appearance remarkably like the spinal cord. On section it was soft, almost diffluent, and no lumen could be detected.[1] Within

[1] The ureter was exhibited by Mr. Targett as a card specimen, before the Pathological Society, 'Path. Trans.,' vol. xliii, 1892, p. 92.

the bladder, the growth protruded from the orifice of the right ureter as a polypoid tumour about the size of a cherry. The bladder was otherwise healthy. Owing to the obstruction of the pelvis of the right kidney that organ had become hydronephrotic, and consisted chiefly of enormously distended calyces. When the specimen had been carefully dissected it was found that the right kidney was entirely free of growth, and that the tumour had grown in between the pelvis of the kidney and the supra-renal body, but was outside the capsule of the kidney.

*Left kidney* was small and moderately granular on the surface. The capsule was adherent, and the cortex wasted. There was some "gravel" in the pelvis.

*Testes.*—The right was white and fibrous, and contained a small gumma. The left testicle and the prostate and vesiculæ seminalis were normal.

*Duodenum and pancreas.*—The head of the pancreas was invaded by growth, which had extended along the duct into the second part of the duodenum, where it had formed a flat ulcer 2 inches in length and 1 inch across. The common, cystic, and hepatic ducts were all moderately dilated, but a stream of water could be forced through them.

*Liver*, 53 oz. Small secondary deposits scattered through it. *Spleen*, 5 oz., normal. *Adrenals* normal.

*Aorta.*—Abdominal aorta completely embedded in growth, narrowed but not obstructed.

*Inferior vena cava.*—Below the right renal vein it passed through the growth, and could not be traced. Both common iliac veins thrombosed. Right renal vein invaded with growth, and its lumen almost blocked.

On microscopic examination the growth proved to be a round-celled sarcoma.

*Remarks.*—The diagnosis of malignant disease of the kidney seemed to be inevitable in this case from the association of hæmaturia with tumour occupying the right lumbar region and adjacent part of the abdomen. And yet the hæmaturia, when carefully inquired into, though showing the implication of the genito-urinary tract, was not of itself evidence that the kidney was the particular part of the tract

concerned, and the post-mortem showed that the organ itself was actually free. The feature about the urine which determined me in believing that the bladder was at least involved as well as the kidney, was the presence of large clots, which caused much pain and distress on being passed through the meatus urinarius. These clots were of such a size that they could not have been formed in the pelvis of the kidney, and if formed there they could not have traversed the canal even of a normal ureter to reach the bladder. The clots were oval in shape, with one margin thick and rounded, and the other irregular and fringed. They measured, as the report states, from ½ inch to 3 or 4 inches in length.

I may perhaps be allowed to dwell on the subject of these clots as diagnostic origin of the hæmorrhage from the bladder, or, at any rate, of blood reaching the bladder in a coagulable condition, although it is a matter that is considered to fall within the province of the surgeon. Still hæmaturia is common ground until the source of the hæmorrhage has been determined, and surgeons will allow that it is very important that physicians should know when they ought to hand over cases into their charge.

The importance of blood-clots is recognised by many surgical writers, but they are ignored by some. The chief point is obviously to distinguish vesical clots from the cylindrical or vermiform coagula, which may form both in the ureter and in the urethra. Thus flatness is allowed to be the main characteristic of clots formed in the bladder, while they are further described as circular, round, roundish, or irregular. Mr. Hilton gives one of the best descriptions of these clots and their value in diagnosis, in one of the excellent clinical lectures delivered by him at Guy's Hospital, and printed in an earlier volume of these Reports.[1]

"With hæmorrhage into the bladder the form of the clots is determined by the shape of the vesical cavity. When first passed from the urethra, such coagula may seem to be cylindrical, so that you are, perhaps, inclined to say they are urethral clots; but put them into a dish of water and they will then resume their flattened, circular, true shape. If the bladder is full of clot, nothing but blood-stained

[1] 'Guy's Hosp. Rep.,' 3rd series, vol. xiii, 1868, p. 27.

serum and urine will perhaps escape for days until the clot is broken up by a catheter. Coagula formed within the bladder are somewhat irregular in outline, mostly circular, flattened masses levelled off and fimbriated. If they have remained long *in situ* their fringed edges are white, being formed of decolorised fibrine. Clots of this description evidently could not have formed in the urethra, nor in the ureter, nor in the pelvis of the kidney; and the blood forming them must have clotted within the bladder itself." He then relates a case in which the diagnosis of a vascular tumour was assisted by a consideration of the clots passed. "I emptied the contents of one of the pots into a basin of water, and had the satisfaction of seeing the clots all come out with their physiognomies and faces well marked and all alike."

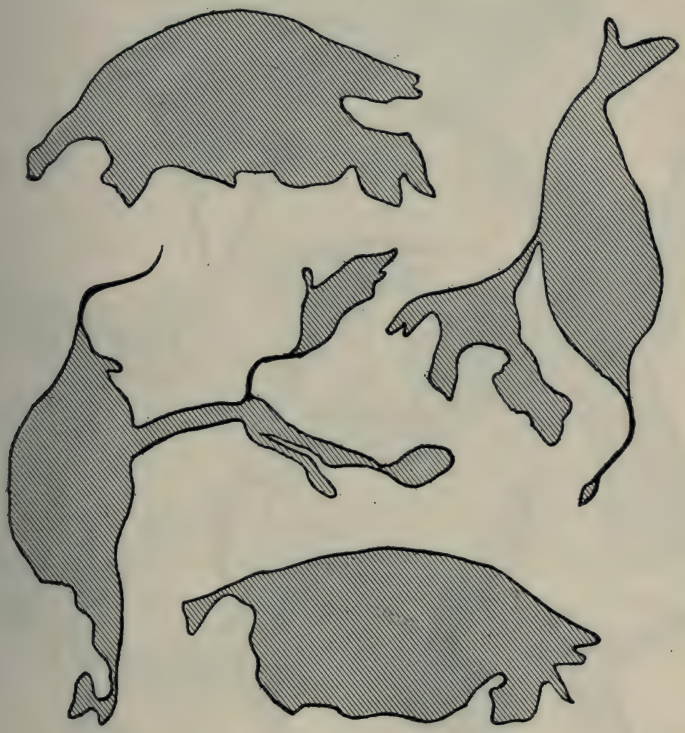
I will very briefly indicate how they are treated by some other writers. "Sometimes they are flat, and are evidently formed on the floor of the bladder" (Fagge); "more or less discoloured, irregular coagula, with fimbriated edges" (Bryant); "large, irregular, shapeless, edge-eroded clots are usually of vesical origin" (Hurry Fenwick); "for the most part roundish or irregular in shape" (W. Coulson); "flat or irregular-shaped clots" (Pearce Gould); "clots coming from the bladder do not present any regular shape; they are short, thick, irregular, sometimes rather elongated" (Albarran, 'Les tumeurs de la vessie,' Paris, 1892). Dr. Dickinson records a case in which they were "of large size, round, pear-shaped, and irregularly elongated and tailed."

In a case of hæmaturia from villous disease of the bladder, which was under the late Dr. Habershon in Guy's Hospital in 1877, these flat clots were frequently passed. The annexed drawings (Figs. 1 and 2) are reproduced from sketches made at the time, partly by myself, and they may be compared with Fig. 3, which represents a clot from the present case. It will be seen that an oval form with pointed ends is frequent; four such clots are represented in Fig. 1; and in Fig. 3 the clot has almost exactly the same form.

To return to my own case, these clots led me to conclude that the blood could not come from the kidney alone, or

that if it did it must come so freely as to pour down the ureter, and coagulate in the bladder; and I sought for evidence that either the ureter or the bladder was invaded. The tumour in the abdomen extended so low down towards

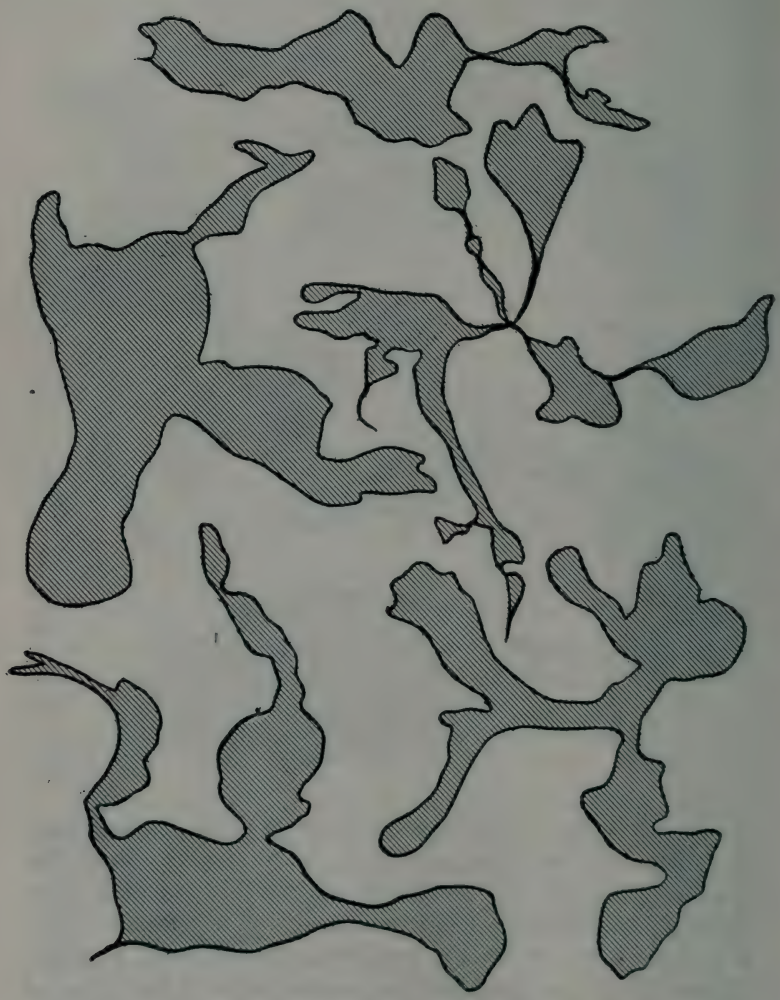
Fig. 1.



the pelvis, that it was quite likely that the ureter was invaded; but on examination of the posterior wall of the bladder *per rectum* I could do no more than feel some doubtful resistance at the extreme end of my finger. The use of a sound or of the cystoscope would have, perhaps, revealed the true source of the hæmorrhage in the mass of cancer which projected into the bladder at the termination of the right ureter; but his condition was so desperate, and the hopelessness of relief so complete, that it was not felt necessary to pursue this clinical point to its extreme. The

formation of a small hydronephrosis by this obstruction of the ureter was also of interest. The lower part of this tube

FIG. 2.



was converted into a round white cord, in which a canal was scarcely visible. By this, of course, the duct was obstructed, and during the development of this obstruction

the pelvis of the kidney thus under pressure must have yielded and dilated into the cavity found. But it is probable

Fig. 3.

that at the time of death the tendency to further dilatation had ceased, since the solid part of the kidney was entirely converted into cancerous tissue, and almost certainly, therefore, there was no further secretion of urine.

The interesting question of the functions of the pancreas might be raised also in this case, since the "head" of that organ was converted into cancer; but the subject was not present to our minds during the patient's lifetime, and no record of the character of the motions appears to have been made.

The usual contrast in the behaviour of arteries and veins to malignant growths, referred to in Case 1, is well illustrated in the present instance.

# NOTE ON

THE

# OPERATION OF CIRCUMCISION.

By N. DAVIES-COLLEY.

The operation of circumcision is so simple that the most meagre details with respect to its performance have been given by the writers of our surgical text-books. It is, however, an operation very frequently performed by the most inexperienced members of our profession; and a great deal of discomfort, and even serious injury, may result from certain mistakes, against which, as it seems to me, our surgical writers have not sufficiently warned their readers.

Mr. Howse, in a previous number of our Reports,[1] has clearly pointed out one evil result which may follow the usual practice of slitting up the mucous membrane of the prepuce along the dorsum of the glans penis, and turning the two halves outwards and downwards, so that the redundant tissue forms a knob beneath the glans, which, in some cases, is almost as big as the glans itself. But there are other evil results which he has not mentioned.

Thus, occasionally, we find that so much skin has been removed that during erection the integuments of the organ are very tightly stretched. One cause of this result is the direction given in one of our text-books that, as the first step of the operation, a dressing forceps should be applied to the penis at the level of the corona glandis. The prepuce is then drawn forwards, with the closed blades of the forceps

[1] Ser. iii, vol. xviii.

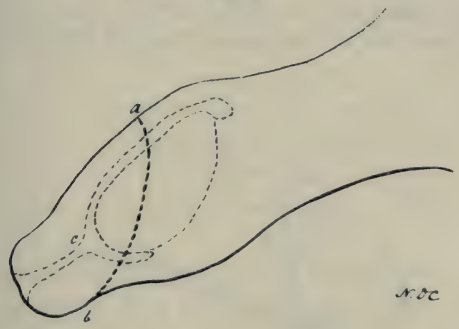
grasping the skin, while the glans is allowed to slip backwards. If, as not unfrequently occurs, the glans penis has already, from the effects of cold, shrunk back into the skin near the root of the penis, the shave of the knife in front of the forceps will take away nearly all the skin of the organ. Another, and perhaps more serious error, is to fail in dividing the mucous membrane of the prepuce as far back as the sulcus behind the glans, and then to leave so much of this mucous membrane behind that it closes up again, so as to produce a still greater constriction in front of the glans than before the operation. I once had a child brought to me who had to pass his urine with great difficulty through an orifice which would hardly admit the point of a pin. He had been circumcised a few months previously, and the operator attributed this obstruction to the neglect of the child's nurse, who had not attended to his injunction that the foreskin should be drawn back every day. The injunction would not have been needed if a sufficient amount of the mucous membrane had been removed in the first operation. Another, but less serious, result of operating in the ordinary fashion is that a triangular surface is often left beneath the glans without any skin to cover it; and the recovery of the patient is retarded while this surface heals over by granulations.

The method I am about to describe is one which I have used for more than fifteen years. I have never seen any description of a similar method, but I cannot say how far the plan is original with myself. It is possible that for some part of it I may be indebted to the late Mr. Cock; but I do not remember to have seen him perform the operation of circumcision, nor do I know of any description of his method of operation.

The patient being under an anæsthetic, an assistant should grasp the foreskin somewhat obliquely with a pair of dressing forceps forwards and downwards, taking care that the glans penis is not included. He should first, however, observe that the penis is not unduly retracted, and the incision in the skin should begin upon the dorsum at a point corresponding to that part of the glans which is half-way between the meatus urinarius and the corona. If the penis is retracted he

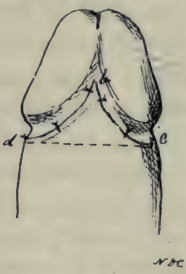
must apply the forceps further forwards. Before closing the forceps, care must of course be taken that the glans penis has been allowed to slip back behind the blades, and that only the foreskin is grasped. The surgeon should now apply the knife in front of the forceps, and cut through the

Fig. 1.




dorsal half of that which is grasped. Next, after removing the forceps, he should cut through the rest of the skin with a pair of scissors downwards and forwards, so as to leave a sharp point in the middle of the under surface of the penis (*vide* figures 1 and 2). (The object of this pointed projec-

Fig. 2.

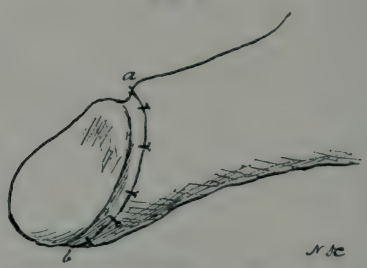



tion is to fill up subsequently the triangular interval which is otherwise left when that portion of the mucous membrane of the prepuce, to which the frænum is attached, is removed.)

The second stage of the operation is to slit up the mucous lining of the prepuce which still envelops the glans penis. This should be done as usual, along the dorsal surface, either with the blunt-pointed scissors, or with a director and knife. The former is preferable, unless the

tissues are so thick as to make it uncertain whether one blade of the scissors has entered the urethra. All adhesions should be torn through with the nail or a probe, until the corona and the sulcus behind it have been fully exposed. The surgeon should then clip away with the scissors all the mucous membrane of the prepuce except a frill, so to speak, a sixth of an inch broad all the way round the corona from the dorsal aspect to the frænum. Usually

Fig. 3.




but little hæmorrhage occurs; but what there is can readily be stopped by twisting the bleeding points with a Spencer Wells' forceps, or by tying a fine catgut ligature around it. The margin of the skin will now be found to fit accurately along the cut edge of the mucous membrane, and it should be attached by fine sutures of silk or catgut, which should be passed by means of an ordinary sewing needle, close to the edges of the skin and mucous membrane, and drawn tightly, so that they do not require to be subsequently removed. The wound should now be sprinkled with iodoform powder, and covered with a film of absorbent cotton-wool, which should be painted over with flexible collodion. This dressing will usually remain on until the wound is healed; but if, on account of the restlessness of the patient, it becomes loose or drops off, a little boracic ointment should be smeared on in its place.

The advantages I claim for this mode of operation are: (1) there is no possibility of recurrence of the phimosis, as the mucous membrane, to which the constriction is due, has been completely taken away; (2) there is never any lump formed behind the frænum by the infiltration of reflected mucous membrane; (3) the skin is left in such a quantity

as to allow erection of the organ to take place without any tension; and (4) the whole of the wound usually heals up by primary union.

In fig. 1 I have indicated the line of incision, *a b*, through the skin, and at *c* the stricture of the mucous membrane, which is the cause of the phimosis; the finer dotted line represents the mucous membrane lining the prepuce and covering the glans penis.

In fig. 2 is shown the adjustment of the pointed process of skin (*b*) in the angle left by the remains of the frænum. The dotted line (*d e*) indicates the edge left to the skin on the under side of the penis by the usual mode of operation, and the triangle (*b d e*) the bare surface which has to heal by granulation.

Fig. 3 represents the penis after the suturing of the edge of skin (*a b*) to the strip of mucous membrane which is left along the back and sides of the corona.

ON THE RESULTS OF

# ONE HUNDRED AND THIRTY CASES OF EXCISION OF THE KNEE.

By H. G. HOWSE, M.S.

## THE ANALYSIS OF THE CASES

By G. NEWTON PITT, M.D.

The following cases were operated upon, partly in Guy's Hospital, partly at the Evelina Hospital for Sick Children, during the years 1873 to 1884. The object of this paper is to show not only the *immediate* results of the operation of excision of the knee, but also, as far as possible, the condition of the limbs many years afterwards. For this purpose none of the cases operated on since 1884 are here published. It is hoped that returns such as these may, in the future, form a basis of comparison between this operation and some of those more lately introduced, both as regards immediate results, and as regards the condition of the patient, with respect both to life and limb, many years afterwards. The earlier cases in the tables represent my first experience of the operation; the results, therefore, cannot be looked

upon as unduly favourable to it. Later on in the paper there will be found a comparison between the first dozen cases operated upon, and the last dozen, showing the great difference in the success obtained. This may be attributed partly to increased experience, and partly to improved appliances.

The operation of excision of the knee is still regarded with disfavour by many surgeons, and in most of the recent text-books the statistics and the conclusions drawn therefrom are based upon operations performed without the use of antiseptics.

Thus in the 'British Medical Journal' for 1880, Mr. Holmes published the results of 245 cases of excision of the knee performed during the five years previous to 1878 in the London Hospitals. There were twenty-one deaths from the operation, and four after amputation. Four cases were unaccounted for, and in forty-seven cases the operation failed, most of these being amputated.

Kocher, at the International Medical Congress in 1881, brought forward sixty-four cases, with six deaths (three from septicæmia, and one each from hæmorrhage, carboluria, and acute tuberculosis).

Mr. Holmes, in his 'System of Surgery' (3rd Edition, vol. iii, page 758), says:—"The acceptance which this operation has met with is not due to its lower rate of mortality. No one, I think, now denies that in similar cases the operation of amputation gives a lower rate of mortality than excision. The higher death-rate, however, only imperfectly represents the ill-success of the operation, for to those who have died we must add those whose limbs have been amputated or have remained useless. . . . There is no conceivable reason why excision should be less fatal than amputation. It is sometimes said that the hæmorrhage is less, but I am far from convinced of this. . . . In excision the operation is often a bloody one (though sometimes not so), and secondary hæmorrhage is by no means rare. . . . The immediate shock, as far as I have seen, is quite as great as in amputation.

"In all cases where sinuses exist, leading for any considerable distance, and generally whenever the disease is of

very long duration, excision should only be undertaken as an exploratory measure, and everything should be in readiness for amputation, if the softened condition of the bones should render the latter operation necessary, as will very probably be the case, even if no part should be found absolutely carious or necrosed."

The statistics about to be given do not bear out these statements. The risk to life, due to the operation, when properly performed, is extremely small. Examination of the mortality table, to be given later on, makes it very doubtful whether the operation of amputation gives a lower rate of mortality than excision. The operation is not a bloody one, and secondary hæmorrhage is almost unknown. Extensive necrosis of the ends of the bones, long sinuses, and a history of the disease having existed for months or even years, by no means preclude the production of a most serviceable limb. In two cases sequestra, 4½ inches long, were removed with a good result ultimately.

Mr. Bryant's statistics in the latest edition of his 'Surgery' (4th edition, vol. ii, p. 514), give the mortality as 22 per cent., and for cases under twenty years of age 27·8 per cent. The corresponding figures, deduced from the following 130 cases, taking all the deaths *from all causes* which have occurred within six months after excision are 6·2 per cent., and for cases under twenty years of age 2·3 per cent. If we exclude the deaths from tubercular meningitis and general tuberculosis, the percentages will, of course, be much more favourable. Of the four deaths occurring within one month of the excision, and which may, therefore, be considered as in some way due to the operation, one (Case 86) was due to uncontrollable vomiting after chloroform and ether (which would certainly have occurred, whatever operation had been performed); another was an accident (Case 4); the third (Case 95) was a man who had previously lost the other leg from pulpy disease, and who was intensely desirous of saving his remaining member; he died more from the condition of his viscera (scrofulous kidney, tubercular intestine, &c.) than from the effects of the operation. The fourth (Case 48) was one which can scarcely be fairly included in a table of deaths due to excision, the whole of

the front of the knee-joint having been literally blown away by the discharge of a gun. The patient was a great drinker, weighed about eighteen stone, and was partly jaundiced at the time of admission. He refused amputation, and the skin was so much destroyed that, even after excision, it could not be brought together, so that a wide gap was left over the ends of the bones. He died rather from the effects of the accident, and from the degenerated condition of his viscera, than from the excision.

Nevertheless, I have added this to the list of fatal cases, because I wished to include all the deaths which occurred *after* excision, whether really due to it or not.

In 'Ashhurst's International Encyclopædia of Surgery' there is a much more accurate appreciation of the value of the operation. Dr. Ashhurst, jun. (vol. iv, p. 518), there records twenty-six excisions in twenty-five cases (one a re-excision) with twenty-two recoveries (one after amputation), two cases still under treatment, and only two deaths. And in a special article on "Excision of the Knee-joint," Dr. Fenwick, of Montreal (vol. iv, pp. 535 to 542), records twenty-eight cases, of which twenty-four were successful; one died from pyæmia, and two required amputation, while one gave a doubtful result.

As many surgeons, however, are still unaware what excellent results can now be obtained by excision of the knee, it has been thought desirable to publish the following cases, which probably represent a larger personal experience than any hitherto recorded. It may be shortly stated that more than half of the 130 cases have most excellent, serviceable limbs; two cases only died from the immediate effects of the operation (both from vomiting within five or six days after it, but one of these had tubercular kidney, lung, and intestine); six cases died from tubercular mischief within three years after the operation; one died from gangrene due to the sound leg being too tightly bandaged; and one died from gun-shot wound, as before described.

The cases were by no means picked ones. Many were children in such miserably feeble health, that from the first it seemed almost inevitable that amputation would ultimately be necessary; but every effort was made to save the leg, as

some of the earlier cases had most unexpectedly improved after the operation, and left the hospital with a sound, well-ankylosed limb. The good results are mainly due to antiseptic dressing, not a single case dying of pyæmia or septicæmia. Other things have, however, conduced to it, as, for example, the mode of fixing the limb, which has been carried out in all the later cases; the treatment of the pulpy material; and the treatment of the sinuses which occasionally persist after the operation.

Out of the 130 cases there were 65, or exactly 50 per cent., in which the results *are known* to be most satisfactory a long period (often many years) after the operation. In these are included eight cases in which the shortening exceeded two inches. In seven of these the shortening was produced by the removal of sequestra, or by the large slice of bone which had to be sawn off to straighten the limb; but in every case the resulting limb was most serviceable, and far superior to a stump. In most of the remaining cases the shortening was less than one inch, and the limb was straight, or, if flexed, only so slightly as not to impair its usefulness. Twenty-one cases have been lost sight of, but in all these the results were good when the patients left the hospital. It will, therefore, not be unreasonable to conclude that the great majority have continued so; hence many of these cases also should be considered successful.

In twelve cases, the ages of which varied from three to twelve years, the leg became flexed at a considerable angle. In ten others there was very slight flexion, but not such as in any way to interfere with the usefulness of the limb.

In twenty-six cases it was ultimately necessary within three years after the excision to remove the limb through the lower third of the thigh, on account of profuse suppuration, or of persistent sinuses which led down to necrosed bone or to pulpy granulations. In two cases the limb was removed on account of lardaceous disease, which then gradually disappeared. In several cases an effort was made to save the limb by excising the knee, and undoubtedly in some of these the wounds would have healed if the children had been in better health. Hence, in discussing the value of the operation, these cases should not be considered as failures, except

we add the proviso that many of them were almost hopeless from the first.

The effort to save the limb does not seem to have materially impaired the patient's chances of life. Most of the cases amputated did well, although the conditions under which the operation was performed seemed very unfavourable for success. Out of twenty-six amputations five only died; two of these from tubercular meningitis and general tuberculosis; one from phthisis two years after the amputation; one from unknown causes fifteen months after the amputation, and one from hæmorrhage.

The question has been raised whether tubercular disease of the system starts into activity on disturbing a local tubercular deposit, such as a pulpy knee; and it is worthy of notice that in the following cases where, at the time of the operation, no general symptoms of tubercular disease were discoverable, they soon blazed into activity, and, curiously enough, in each case after an interval of three weeks.

Case 5, æt. 21 years. Hæmoptysis three weeks after the operation. Death from phthisis in two years.

Case 11, æt. 21 years. Tubercular meningitis came on three weeks after the excision; fatal three weeks later.

Case 119, æt. 39 years. Three weeks after operation cough and aphonia; leg amputated after two months. Death from general tuberculosis three months later.

While examining into the results of the cases given in the tables at the end of this paper, the question which naturally suggests itself is the following:

When should excision be performed?

It is manifest that in a very large proportion of the cases contained in the tables, the disorganisation of the joint had advanced to such an extent that the performance of *some* operation was absolutely necessary. When grating exists in the articulation, when the cartilages are flaking off the articular ends of the bones, when caseous osteitis and possibly sequestra exist in the epiphyses, when there is great infiltration of pulpy material into all the surrounding parts, and when there is very extensive suppuration, nearly all

surgeons are agreed that operative interference of some sort is called for. But a large section of the profession still contend that in such cases amputation is the best operation. These tables, however, go far to show that (though certain extremely bad cases should still be amputated without recourse to any intermediate operation) there are very numerous cases in which a useful limb may be saved for the patient by resection.

The conditions calling for immediate amputation may be divided into *constitutional* and *local*.

The *constitutional* conditions are:

($\alpha$) Lardaceous disease.

($\beta$) Tubercular disease of the lungs or other viscus.

($\gamma$) Great emaciation without any very evident visceral disease.

($\delta$) Multiple joint disease.

In the last group removal of one limb will often so far improve the health as to make resection a feasible and successful operation for the other joint. Case No. 25 is a striking example of this.

The *local* conditions calling for immediate amputation are not so well defined. They are:

($\alpha$) The existence of osteitis or periostitis extending far up the shafts of either femur or tibia, as shown by great thickening or tenderness of the bone.

($\beta$) Very great infiltration of pulpy material into the soft parts, extending far beyond the limits of the joints; often associated with comparatively little evidence of any correspondingly great affection of the proper joint tissues.

These cases require the greatest consideration. They are both often associated with the constitutional states (given above) which necessitate amputation. When they are not, it must be remembered that great tenderness and thickening of the bone may be produced by a sequestrum, which may be successfully removed, and later on a useful limb may be obtained for the patient by excision of the joint (see Case No 100). Again, very considerable purely pulpy infiltration may also exist prior to the excision, and yet finally disappear under the influence of the complete rest accorded by this operation to all the affected parts (see Case No. 112).

This diffuse pulpy infiltration is, however, of distinctly evil import in the prognosis—of much worse import than has hitherto usually been recognised.

I have thus attempted to define the group of cases on which amputation is called for. At the other extremity of the series are the cases which should be treated by rest alone. And the principal difficulty met with by all surgeons consists in defining when the treatment by rest should cease, and the more active treatment of the joint by excision should be undertaken. I am very much inclined to think that a *time limit*, as regards the duration of the disease, will be found a most valuable aid in forming an opinion as regards what ought to be done. This test, of course, only applies to the cases of chronic joint disease, and not to the more acute disorganisations, such as occur in pyæmia, for example. By examination of the tables it will be found that in nearly all the earlier cases, the disease had lasted a very long time previous to excision. Indeed, in all these cases I started with the determination that, unless very urgently called for by the acute disorganisation of the joint, I would not excise any joint which had been less than a twelvemonth under treatment either by myself or by others. It will be seen that in the majority of the cases the disease had lasted for a much longer period. As my experience of the value and safety of the operation increased, and I became more convinced of the hopelessness of obtaining a useful joint in any case of well-marked pulpy disease, I thought it necessary to diminish this limit, and am now inclined to support the dictum that *when a well-marked case of pulpy disease has lasted over six months, it is not worth while, in the interests of the patient, to attempt the conservation of the joint for a longer period.*

In saying this, I do not wish to advise the excision of *every* joint which has been six months affected, any more than I wish to say that no joint should be excised unless it has been for six months affected. The dictum essentially depends upon reasonings connected with the state of the parts after the disease has been running a six months' course.

The *diagnosis*, in fact, of the exact state of the interior of the knee-joint at any one period of pulpy disease is always

a matter of difficulty. Such joints come before the surgeon swollen, with the normal bony outlines more or less lost, and the parts more or less painful on pressure. The question then always arises: What is the exact pathological state of the various tissue forming the joint? Has the mischief commenced in the synovial membrane, or in the bone? If in the former, is it still mainly confined to the synovial membrane, only causing partial erosion of the marginal portions of the cartilages, or has it secondarily extended to the bone? If in the latter, is the epiphysis only infiltrated with pulpy patches, or is the bone in a condition of eburnating osteitis? Are such patches of hardened, inflamed bone forming definite sequestra, surrounded by walls of granulating inflamed bone, or are they unseparated—still continuous with the surrounding osseous tissue? Finally, has the diseased bone caused perforation of the overlying articular cartilage, and to what extent has it caused the articular cartilages, synovial membrane, and the ligaments of the joint to be secondarily implicated?

In each case such questions, which must necessarily present themselves to the mind of the surgeon, are very difficult to answer. I do not think there is any perfectly satisfactory answer to the question which must be asked first of all, viz. has the disease started primarily in the bone, or in the synovial membrane and capsule of the joint? —unless the surgeon has been watching and following the case from the very first. Cases of primary epiphysial osteitis are more apt to follow a local injury to the joint; they are, in my experience, much more rare than the other form, and the exact symptoms caused are much more obscure. But they are more apt to cause local suppuration than the other form (Case No. 99 is an example in point). If, on the other hand, the patient is decidedly of a scrofulous type, and has had one or two attacks of swollen painful joint, the first attack being, perhaps, apparently an ordinary case of synovial effusion from over-walking or over-running; if, however, this swelling has never *entirely* disappeared, but has left a thickened outline round the joint, more especially apparent at the sides of the pouch of synovial membrane under the quadriceps extensor; if, during the first six months there

has been a tendency to repetition of the primary attack once or twice, leaving the joint swollen, white and shining, painful on pressure, with nocturnal starting pain, then I think, although the rule is not absolute, that we are justified in concluding that the disease has started as a capsulitis or synovitis.

It is to such cases, which form by far the greater number of those we are called on to treat, that these remarks principally apply. The question here becomes of importance—When are we to excise, and in what cases to continue the treatment by rest? And it is in these that I think the importance of considering the *duration* of the malady is very frequently over-looked. It must be remembered that pulpy disease is always an actively destructive process on the joint tissues. The capsular and crucial ligaments inflame, vascularise, and soften under its influence, so that they are unable any longer to bind the bones firmly together. The semilunar cartilages ultimately vascularise and soften, thus losing their familiar outline and consistence, so that during the operation it is sometimes difficult to say, except from position, what is semilunar cartilage and what is not. Pulpy tissue erodes and finally absorbs the articular cartilage, and so causes its disappearance. It is true that in a case which undergoes the so-called "natural cure" these tissues are replaced by a form of connective tissue. But how much less efficient the new tissue is than the old joint tissue everyone who has had the opportunity of comparing one of the so-called "cured" pulpy knees with the healthy joint must have had the opportunity of observing. If we examine the material microscopically the reason of its destructive power is soon seen. Masses of cells lie longitudinally between the fibres of which the ligaments are composed, separating them from one another and destroying the strength which lies in their fasciculated arrangement. These cells, from their rapid growth and increase in number, tend to death from deficient blood-supply, and hence to caseation and necrosis. Thus it is that in such cases we get abscesses containing masses of caseating material.

Pulpy disease is, therefore, essentially a *destructive* disease as far as the joint tissues are concerned. Every step in its

progress upon a healthy joint is marked by some healthy tissue damaged, or, in the case of the cartilages, destroyed, so that it cannot be replaced. But pulpy disease, from a change in the surrounding conditions, may be arrested at any stage. It therefore becomes important to consider how far it may advance, and yet, if stopped, the new tissue produced may efficiently replace the old. I think it must be admitted that when capsulitis (which must always be conjoined with more or less thickening and inflammation of the synovial membrane) *alone* exists, the new material produced by rest and time very nearly equals the old in strength. And yet a joint which has suffered one or two attacks of pulpy capsulitis very rarely equals in strength a healthy one, even when it has undergone the most complete "natural cure." The reason of this is not difficult to find. All ligaments which are inflamed become softened, and lose their resisting power. Any slight strain put upon the crucial and lateral ligaments in this state of chronic inflammation tends to stretch them. Though the stretched ligaments may have some power of recovery, if the process be not too far advanced, yet such recovery very frequently does not take place, and hence the *loose-jointed* cases one not infrequently sees after an attack of capsulitis. When we remember how frequently children amongst the poor continue to use their limbs until the increasing pain makes it impossible to do so any longer, when we remember the leverage exerted upon the ligaments of the knee-joint, and that the bones are held in contact by scarcely anything more than the ligaments, it is not to be wondered at that they so frequently stretch—in most cases beyond recovery. It is probably from this reason that cases of this loose-jointed condition are less frequently seen amongst the rich, who can afford to provide and attend to the necessary appliances for the treatment of such a state sufficiently early.

This loose-jointed condition is at the bottom of a good deal of the trouble and risk which such cases run. (A good example of it is seen in Case No. 12, which was recorded in 'Guy's Hospital Reports,' ser. iii, vol. xx, p. 531). If the apposition of the joint surfaces be not properly provided for,

by the right degree of tension of the ligaments, awkward movements of one bone upon the other are very apt to occur; perhaps catching in the movement some part of an over-sensitive semilunar cartilage, and squeezing it between the bones. This may bring on a sudden intense pain in the joint, which may cause the limb to give way and the patient to fall. It is pain of the same character as that described by patients with "loose cartilage." Given a patient of scrofulous type, who has had one attack of pulpy capsulitis, we have in such a state, and its resulting accidents, quite sufficient cause for a repetition of the attacks of general pulpy arthritis. This is a reason why one attack of pulpy capsulitis so frequently leads to another. Even if it does not, the loose-jointed condition is always one which inconveniences the patients not a little for the rest of their lives. It is always an insecure joint, and necessitates the patient wearing some support. Hence such patients nearly always have recourse to elastic knee-caps, Martin's elastic bandages, and often to appliances of a more substantial nature than these, the effects of all of which are to re-inforce the support given by the weakened ligaments, and to limit the movements of the joint, so as to prevent the extreme and sudden movements, in which most frequently the danger to the patient lies.[1]

If such accidents as these tend to follow an attack of

[1] The loose-jointed condition sometimes prevails in more than one joint in patients of scrofulous diathesis. Thus, in a private patient of my own—a young lady æt. 20—both knees and ankles were weak from this condition, and she constantly wore elastic supports, with the assistance of which she was fairly comfortable in these joints. She came under my care for what was substantially the same condition in the left hip-joint. This manifested itself by pain and occasional subacute attacks of synovitis, more especially after much dancing or long exertion. There was a certain unevenness of movement in the joint, which the patient called "grating," but it was most probably due to irregularity in the surface of the cartilage; it was certainly not bone-grating. After a lengthened period of treatment by rest and leathern splints I devised for her a strong elastic support, which gave very considerable relief, but she got impatient at not getting entirely well, and disappeared. I heard some considerable time afterwards from her that her hip-joint had been subsequently excised; the result of the operation was not, however, entirely satisfactory. This patient's father suffered from an osteo-arthritic condition of many joints, evidently of constitutional origin. The condition had come on gradually, and he had so far got used to it that it did not call for other than medicinal treatment at my

pulpy capsulitis, *i. e.* the simplest form of pulpy disease, it must be manifest that, where the destruction is more complete, the effects will be much more lasting and disastrous to the patients. If the patient is subjected to surgical treatment, and the joint is kept in a state of absolute rest in the extended position *from the very commencement* of the first attack of pulpy capsulitis, the patient *may* get well without material damage to the articulation. But how often are we obliged to treat joints, especially among the children of the poor, which have not had this attention. And if from any cause the treatment by rest and immobilisation does not arrest the disease, its spread to the other structures of the joint is only a question of *time*. The crucial ligaments will become affected, the semilunar cartilages will become vascularised and softened, and the articular cartilages eroded at all such points as may be in contact with the rapidly extending pulpy portions of the synovial membrane. It must be manifest that when these changes have fairly commenced, the integrity of the joint, *as a joint*, is gone. The question then is, not whether by rest this joint may return to its normal condition, but what can be the most favourable state it can assume, should arrest of the disease at any time occur. In a few very exceptional cases nature conserves, even in this condition, a useful limb for the patient; but I think any impartial observer, who will compare them with excised knee-joints, will admit that the resulting limb is far inferior in solidity, painlessness, and general usefulness.

But it is in very few cases that arrest of the disease takes place at this stage. It generally goes further—to complete destruction of the joint—even where the most elaborate appliances and best surgical treatment have been used. When the disease has become so general as to affect the crucial ligaments and the semilunar cartilages, and to erode the articular cartilages, it nearly always affects also the bone, and this introduces a new factor in the case, and one much more likely than anything else to cause suppura-

hands. I have seen many other examples of this loose-jointed condition both in private and hospital practice, and especially among the patients applying for treatment at the National Truss Society, where elastic support is the favourite means of obtaining relief in this condition.

tion and complete disorganisation of the joint. For any long-continued epiphysial osteitis results almost invariably in granulations forming between the articular cartilage in the bone. These first absorb the attached surface of the articular cartilage, eating out little pits therein, and then cause it to detach in pieces, which become free in the pus in the joint, forming the condition known as "flaking of the articular cartilages." For a spontaneous cure to take place in this condition, it is necessary either that the pus and the flakes of cartilages (which are often of considerable size) should be got rid of by being discharged through the opening of an abscess, or else that they should become encapsuled, when they generally partly absorb exceedingly slowly, and partly form the cheesy products of a caseous abscess in the centre of the capsule. The exceeding slowness of these changes may be estimated when I say that in excising a joint I have seen the flakes of cartilage almost unaltered many months, or even as much as a year or two, after the date of their probable formation. Such a fact as this will tend to show how very slow the complete consolidation of a pulpy joint in this stage must necessarily be, even under the most favourable conditions.

The most common way, however, for spontaneous cure to take place, when a joint has reached this stage, is through abscess-formation and complete destruction of the joint. The final result of this is very similar to what is found after excision. All the articular cartilage gets thrown off and absorbed, the semilunar cartilages become vascularised and adherent on either side to the granulations springing from the inflamed bone, until nothing is left but a mass of granulation-tissue between the bones, which gradually changes into fibroid connective-tissue and finally ossifies. This process does not generally go on simultaneously over the whole joint surface, but spreads from circumference to centre. Hence it is that if arrest of the process of ankylosis at any time take place, the central part over each condyle of the femur or tuberosity of the tibia is frequently found to present some unaltered remains of the original tissue of the joint. Should such arrest occur, the result is fibrous ankylosis with probably a slight (*very* small) amount of movement about the joint. Such movement is very generally

absolutely prejudicial to the patient, as it contributes to the sense of insecurity of the limb. And if, by any accident, the movement be extended over a greater range than allowed for by the adhesions, it tends to strain them and to light up again an attack of acute inflammation of the part.

The inflammation of the bone again not infrequently runs a more complicated course than that represented above. It may inflame in patches, undergoing the process of eburnating osteitis. Such patches of bone not seldom necrose, and form sequestra in the interior of the epiphysis, the cavities containing which nearly always communicate with the remains of the joint cavity. When this process occurs, it may safely be said that a complete natural cure never takes place until the sequestra have been got rid of. Very numerous examples of it will be found in the tables (Cases Nos. 13, 20, 22, 25, 29, 30, &c.).

The *length of time* which this process of natural cure takes in any case is a most important subject for consideration. Surgeons who are opposed to the practice of excision generally give a period of two years for the process of ankylosis to complete itself. This is generally far too low an estimate. The details of very many of the cases in the tables will show how many years the disease had been slowly progressing before excision was had recourse to, and how very slight the progress towards natural cure was at the time of the operation. It may be objected to these cases that they principally occurred in poor people in whom the surgical treatment (by appliances, &c.) was deficient. This is true, but as far as my experience goes, even among the wealthy in private practice, the two years estimate is one very much below the mark. Thus in 1878 I saw in consultation a lad, aged eight years, for whom I had advised excision. The disease had then lasted more than two years, and had been seen by myself at intervals nearly the whole time. Every surgical appliance was at hand, but the two years had not nearly sufficed for the consolidation of the joint. The surgeon with whom I consulted was adverse to the operation, and the usual advice was given that complete rest for two years more would probably result in cure. About two years and a half subsequent to the consultation he was brought to me again,

having been continuously under treatment in the interval by a general practitioner. Pus was then forming in connection with the knee-joint, which had become slightly flexed and was far from being completely consolidated. The history in the interim had been that all pain in the knee had ceased in from six to nine months after the consultation, rest being strictly enjoined; that after this he had been six weeks at Margate, and then had gone to school, walking with crutches, and with a leathern case on the knee, and that the present suppuration had been only a short time in forming. I recommended absolute rest again, and a fresh leathern case to fit the exact bend of the knee. I did not again hear of him till December, 1885, when I learnt from his mother that with complete rest the abscess receded, and after six weeks he had but little pain in the knee. The letter also stated that "for about a year he complained occasionally of soreness and slight pain, but the attacks were always of a mild character. He wore the splint for about two years after we last saw you, walking at the same time on crutches. During that period the limb wore a very shrunken appearance, being not more than half the girth of the other both above and below the joint. He discarded his splint and crutches about the same time, and the limb has gradually grown to the same size as the other. It is now to all appearance perfectly sound, and his exercise is not in any way interfered with. The leg is rather more bent than when you last saw it. . . . . Of late it has seemed perfectly fixed. It is, perhaps, three or four inches shorter than the other. . . . He generally uses a stick in walking, and in this way can walk eight or ten miles with no more than ordinary fatigue. About half that distance would probably be enough for him without the help of the stick. During the last two years he has done pretty much the same school work as is usual with boys. But previous to that he was allowed to take his education very easily."

In this case then, though it was one so favourable in its surroundings and in the general condition of the child's health, that a two-years' duration of the treatment was unhesitatingly given by one of the ablest and most eminent surgeons of the day, we find that two and a half years after-

wards consolidation was not nearly complete, and that matter was beginning to form about the knee. It required two years more before he was able to dispense with the splint and crutches, and even then the leg is left so much bent, and so much shorter than the other, that he walks habitually with a stick. It is probable, however, that some of the shortening may be ascribed to the flexion, and not to want of growth of the bones. The case therefore has lasted under treatment for over seven years, during the whole of which period his education has been most materially interfered with. Finally, the result, at the present time, is not such as to warrant the belief that we have absolutely seen the end of the case. Somewhere about the joint there is probably encapsuled the caseous remains of the abscess, which may yet cause trouble.

The following case, which is that of a personal friend, bears equally forcibly upon the duration of time of these maladies. It is probably not a pulpy case, strictly speaking, but the result of a more acute arthritis following an injury. The disorganising changes have been, however, similar, and as they are not the result of a profound strumous cachexia, they will all the more forcibly speak for the slowness of the changes which lead to consolidation.

T. L. W—, æt. 47, a barrister, struck his left knee when ten years of age. A few days afterwards a boy accidentally struck it again on the same place with a stone. This caused severe pain, followed by an attack of acute inflammation in the joint with but slight swelling. He lived at this time in a healthy country district in Ireland, and was under the care of a local practitioner, who kept the joint at rest, blistered, and leeched it. But apparently no efficient splint was applied, for the limb became flexed to a right angle. Amputation was then proposed, before consenting to which his father took him to see a consulting surgeon in Dublin. Here a splint was applied with a screw-extension arrangement, and the friends were advised to extend the leg by means of the screw to a certain degree every day. It was believed the extension was carried out a little too fast, and backward dislocation of the tibia and fibula took place. No

harm, however, resulted from this, except that it probably delayed the cure. Consolidation went steadily forwards, but it was more than four years before he was able to throw away his crutches and sticks, and walk without support. The present state of the joint is—partial backward dislocation and outward rotation of the leg; slight flexion (*i. e.* to an angle of 165°); complete ankylosis of the joint in this position; shortening of the limb to the extent of rather over 2 inches; well-marked pes cavus, and the heel slightly raised from the ground. The shortening is due partly to want of development of the limb, and partly to backward dislocation, and slight flexion of the leg on the thigh. He is a broad-chested, well-developed man, of good physique, 5 feet 8½ inches in height. He says that his accident is responsible for his shortness, as he is the first of his race for many generations who has not grown over 6 feet high. The limb is in all other respects, except those above noted, as if the joint had been excised, and he can use it well, walking along plain ground with only a very slight limp.

Here we have a case, as before said, probably not of scrofulous origin, placed in all favourable surroundings, and yet requiring more than four years for complete consolidation. Nobody can look at this leg without seeing how much more favourable a result would be produced now by excision. The backward displacement and consequent unfavourable position in which ankylosis has occurred would have been prevented, and the duration of treatment would have been shortened to between three and six months. In a growing lad, whose school education is materially interfered with by such prolonged surgical treatment, to say nothing of the expense which such treatment involves, any means by which it may be cut short, especially means which produce a better result at less risk, will be welcomed by all surgeons.

Cases such as the two just cited are among the most favourable results of the prolonged treatment by rest. But it may be questioned how frequently such results occur. What is the after-history of cases of pulpy disease of the knee and other joints treated by rest? In the tables we have tried to show what is the after-history of excised

knees, by obtaining the latest information about them, and the result is certainly exceedingly favourable. But no similar experience exists, as far as my knowledge goes, of cases treated entirely by rest, with which these might be compared. We require to know what is the state of patients ten or twenty years after they have been treated for pulpy joints. How many die while waiting for possible ankylosis? Of those who survive, how many live ten or twenty years without the joint again breaking down? How many run the gauntlet of tubercle safely, while awaiting the absorption of caseating products in the slow consolidation of a joint? We fear investigation would prove an alarming mortality amongst such cases—much greater than that obtained when all the parts have been firmly consolidated once for all by excision. But on this point we have not been able to obtain reliable and definite data. The operation is rarely performed, except among hospital patients; and yet, if, instead of years of invalidism with often an early tubercular grave, we can offer our wealthier patients the probability that, after an operation of no great danger and some three months in bed, they will probably possess a firm, sound, stiff leg, which will most likely never be a source of trouble again, we surely can no longer hesitate to recommend the latter alternative.

In the treatment by rest of the pulpy cases the length of time for consolidation may be set down at the shortest as two years. But if we examine the tables we shall see that that period in hospital cases at any rate is very often far exceeded. Thus (*vide* Table II) out of the 130 cases the duration of the malady previous to excision is recorded in 128. In 13 it was under six months; in 16 it was from six months to a year; in 24 it was from one to two years; in 40 it was from two to five years; in 26 it was from five to ten years; and in 8 it was from ten to twenty years.

Thus we see that in 74 cases of the 130, more than half, the malady had already taken the full time usually given for consolidation, and in the great bulk of the seventy-four had very considerably exceeded it. Yet in these cases, at the time of the excision, the malady showed no signs of approaching the natural cure. In most indeed it was simply

a question between excision and amputation, so that the successful excision proved an unmixed advantage to the patients.

In twenty-four of the remaining cases the malady had lasted from one to two years before operation, and in only thirteen cases had it been less than six months, or twenty-nine cases altogether, in which it was below a twelvemonth.

This statistical enquiry into the duration of the malady previous to excision must prove conclusively:

(1) That no undue hurry has been used in adopting the operation.

(2) That the length of time which the natural changes take in effecting a cure in an ordinary case of pulpy disease is very much greater than that ordinarily stated.

The shortening of this period, and the avoidance of inevitable risks during its continuance, is the object which we have in view in excising a joint.

In all those cases in which excision was performed under a six-months' duration of the disease, it was because the malady was so acute as to give rise to grave apprehensions for the safety of the limb if the operation had been delayed longer. It will be noted also that in many of those, in which the disease had a very long duration, sequestra and caseous masses were found in the interior of the joint, which were almost impossible of absorption or removal by any prolongation of the period of treatment by rest. (*Vide* Cases 7, 13, 14, 22, 29, 30, 31, 32, 39, &c. &c.)

In answer therefore to the question: "In what cases should excision be performed?" we should say—

($\alpha$) In all cases in which the disease has advanced so far as to cause flaking of the articular cartilage, and grating in the movement of the joint, whether suppuration be evidently present or not.

($\beta$) Cases in which the softening of the ligaments has extended so far as to give rise to backward displacement of the tibia.

($\gamma$) All cases of over six months' duration, in which there is reason to believe that the disease has started in an epiphysial osteitis, especially if there is evidence to point to the osteitis having given rise secondarily to changes in the cartilage.

(δ) Cases of extensive suppuration in the knee-joint. (All cases of this description which have started from pulpy mischief should be excised. In suppuration which is pyæmic in its origin, incision should be first practised, and excision only after evidence has been obtained of damage to the cartilages, or such damage to the ligaments as to have caused displacement.)

(ε) Cases in which the pulpy infiltration of the synovial membrane has advanced to any considerable degree over the articular cartilage.

(ζ) Cases in which pulpy infiltration has extended beyond the capsular ligament to the crucial ligaments and semilunar cartilages.[1]

The difficulties in diagnosis will be principally felt in ε and ζ, and it is in these that the time limit of six months will be found of most value. In the other headings the conditions requiring excision are so obvious that no mistake can well be made. But if we have only general swelling and pain with loss of the joint outlines, it is not so easy to arrive at an exact determination of the state of the interior of the joint. If then in these cases the pain and swelling continue under treatment without material amendment for six months, I think, even while admitting that in such cases the pulpy infiltration *may* become stationary at any time, that we best consult the patient's interests by excising the joint on these grounds—

(1) That the chances are so very much in favour of the continued progress of the disease.

(2) That even if the disease does not progress it will

[1] Other more rare conditions in which the knee-joint has been excised, and which will be found in the tables, are:—

(η) Osteo-arthritis, leading on to disorganisation.

(θ) Infantile paralysis, with complete loss of power in the extensors of the leg, so that the patient had never walked; muscular power over the hip-joint, however, being retained.

(ι) One or two very extreme cases of genu valgum, conjoined with talipes and more or less infantile paralysis, in which the grouping of the affected muscles was not so distinctly defined as in group (θ).

(κ) Disorganisation of joint due to true hydatid (*Echinococcus hominis*). (One case.)

(λ) Gunshot wound of knee.

These cases were, however, so few in number, compared to the great bulk of the pulpy cases, that they would have to be discussed separately; they cannot be treated of in this paper.

leave a damaged weakened joint, very liable to outbreaks of trouble.

(3) That by means of the operation the duration of the treatment is so very much shortened,—reducing to a few months what would otherwise take as many years.

(4) And, finally, because, by means of it, we greatly reduce the risk of tubercular infection, which results from the absorption of caseating products.

## I. Mode of Operation.

*First, as regards the treatment of the patient immediately previous to excision.*—The only point I wish to raise here relates to the advisability of straightening, as far as possible before operation, a flexed knee-joint. Very many of the cases (especially among hospital patients, where the use of splints or other surgical appliances has not been carried out satisfactorily) come into the wards with the leg flexed to a right angle, or even more. It is more especially in these that I wish to lay stress on the importance of a previous straightening of the limb. For the success of the operation and the ultimate success of the case depend in great measure upon it. It may be done either by weights applied to the leg through the agency of extension-strapping, or else by a splint with the usual screw-extension arrangement. By a continuance of steady, though not very forcible, extension even the worst looking cases may be very greatly straightened previous to the operation. Every inch gained in this way helps most materially in the success of the case. If it be not done, the operator will have to do it at the time of the excision, and in several cases I have seen very considerable force applied, and yet the surgeon has been obliged to put up the limb at an angle, and to trust that it might be straightened later on by means of pressure. All such attempts subsequent to excision I regard as risky in the extreme, and fraught with danger to the patient. The bones ought to lie after the operation in a straight line, easily and even somewhat loosely, and no material pressure should be put upon the tissues posterior to the joint. Where this is not attended to, the risks of displacement of the bones subsequently are greatly increased. It is not

displacement alone which is to be feared. In two cases (not in my own practice), in which very considerable force was applied to extend the leg at the time of the operation, gangrene of the limb took place in one, and tetanus in the other. Though other explanations may be given for these accidents, I believe they may best be accounted for by the stretching of the popliteal artery in the one case, and by the stretching of the popliteal nerves, and consequent irritation and damage to them in the other, in conjunction with some failure in the antiseptic arrangements. When an elastic tube, as a bit of india-rubber tubing for instance, is stretched out longitudinally, the lumen of the tube is diminished to a degree corresponding to the stretching. The same thing takes place with the popliteal artery when the limb is forcibly extended; the artery must be drawn out longitudinally, its calibre diminished, and hence the amount of blood supplied to the limb much lessened. It may be asked—why then does it not take place in the forcible extension under chloroform, which is an every-day plan of treatment of contracted joints? But here the conditions are different. Over-extension is prevented by the strong fascial adhesions posterior to the joint. After excision these are in a measure cut away and destroyed; the extension force comes then much more directly upon the artery and nerves. Moreover, in the excised knee the anastomosing circle of arteries all round the joint are in great measure cut, and hence the supply of blood to the limb below the joint is not available, whereas it remains intact in forcible extension without incision. Any material lessening of the supply of blood sent to the limb may in a scrofulous patient, where the artery at best is often of small size, suffice to determine the occurrence of gangrene, especially when we remember the constrained position in which the limb must necessarily lie after the operation. There are other objections to forcible extension at the time of the operation. In the very first case in which I excised the knee-joint (Case No. 1), I had an exceedingly contracted joint to deal with. I tried at the operation to get the limb straight by cutting away enough to enable the bones to lie easily in a right line; but I got frightened at the

amount necessary for this purpose, and after removing several slices, I contented myself with forcible extension for the remainder. In the course of the week following the operation the tibia slipped away from the end of the femur (*i. e.* again dislocated itself backwards), and the lower end of the femur ultimately necrosed and came through the skin, through which I afterwards removed it, as one would the sequestrum from the stump of an amputation. The bones ultimately united and ankylosed in their new position, and the case did well, but the accident caused very great delay in the healing of the case, and is the reason why this patient remained in hospital after the operation nearly a year. It is true that in this case we were dealing with serious mechanical disadvantages in the treatment of our patient—not the least of which was the old box-splint, which it was the fashion at that time to use. But granting all this, the evils of forcible extension at the time of the excision left such an impression on my mind that I would not willingly repeat it. The pain and sleeplessness it causes to the patient, the risks of serious inflammatory infiltration in the stretched tissue posterior to the joint, the possibility of the bones slipping, and the necessity for tight bandaging over the wound to prevent such slipping, all add to the inconvenience of the patient, and tend to militate against success in an operation in which the progress of repair must necessarily depend upon there being as little irritation about the wound as possible. Indeed, the evils of forcible extension at the time of the operation, both in my own cases and in those of other surgeons, have so impressed me that, *in adults*, if there be backward dislocation, so that previous extension is found impossible, or if the disorganisation has advanced so far that partial ankylosis has occurred, necessitating the operation known as "excision *en bloc*," it is better practice to cut away the bone until the tibia and femur lie in a right line with each other freely and easily without strain upon any of the soft parts; even if this necessitates taking away the whole epiphysis, rather than put any forcible extension upon the tissues posterior to the joint. Most of the bone should be taken from the femur. It is not desirable to take

away more than one or two thin slices from the tibia, for if we go lower than half an inch, we shall probably trench upon the superior tibio-fibular articulation, and this introduces a complication which it is desirable to avoid. If this articulation is only partially cut away, it may cause long and troublesome suppuration while it is obliterating itself. If wholly cut away, it will be found that the upper end of the fibula does not readily unite to the tibia; thus the solidity of the tibio-fibular articulation (inferior) is injured, and is a cause of a painful condition of the ankle-joint occasionally seen after excision of the knee. On the other hand, the femur may be cut away to a very considerable extent in the adult above the epiphysis. The femur narrows only gradually above the epiphysis, and affords a sufficiently broad basis of support and union with the tibia to justify this being done with success. The *amount of bone* which may thus occasionally be removed is very great. In Case 1 (already alluded to) over 4 inches were cut away, and in Case 27, $4\frac{3}{4}$ inches were removed, as measured afterwards upon the pieces. Supposing we even give $\frac{3}{4}$ inch of this to the tibia, it would leave 4 inches sliced away from the femur. Both these were very extreme cases. They were both cases of old-standing disease, in which complete backward dislocation had taken place. And I think that in both there was overgrowth of the femur, after the tibia had dislocated itself backwards. This overgrowth is not an uncommon thing, where there is complete backward dislocation and great flexion, especially if it has occurred in youth during the growing period of the bone. It may partly be attributed to the release of pressure on the articular surfaces of the condyles, partly to the union of the tibia in its new position on the posterior aspect of the femur, which (position) very nearly corresponds to that of the growth-lamella between the epiphysis and diaphysis. The vascularisation of all the surrounding parts set up by this attempted union determines a larger flow of blood to the growth lamella, and this causes increased bone-production, and thus lengthening of the femur. Hence the large amount of bone cut away in these cases does not necessarily mean an equal amount of shortening of the limb, the fear

of which I have sometimes seen applied as an argument against excision, and in favour of amputation. In fact, in both these cases, though performed under difficulties, a very useful limb (in Case 27 especially) resulted to the patients.

If then it is important in the adult to straighten the limb prior to excision, to avoid cutting away very much bone, it will be found that the arguments apply with tenfold force in the child. Granting that it is of the greatest importance, for the safety and success of the case, that the bones should lie easily in contact with each other without any considerable strain upon the soft parts, it will be found necessary, when a strongly-flexed limb in a child is being excised, to take away slice after slice until generally the growth-lamella is trenched upon—sometimes the tibial, sometimes the femoral lamella suffering, often both, especially if the flexion is great, and there is any backward displacement. The consequences of damaging or taking away the growth-lamella are disastrous. The limb subsequently, however good the union, does not grow *pari passu* with the other, and the result in adult age is a limb much shortened and disfigured. I have on sundry occasions seen limbs such as these pointed to as a result of excision, and made use of as an argument against the operation, when, in fact, they are only illustrative of defects in the surgeon's mode of operating.

The *methods of extension* which we employ are the ordinary ones, viz. a weight applied by strapping to the leg, and screw extension by a back splint. As additional means I have found benefit from a steaming arrangement applied to one of my cases by a former dresser, Mr. W. Hodgson, now of Exmouth. In the case to which he applied it, the whole limb was wrapped in mackintosh, and steam from a small boiler heated by a spirit lamp conducted under it, so that the limb was kept in a constant vapour-bath for some considerable time (two or three weeks), care being taken to avoid any scalding of the surface from the steam-jet playing accidentally upon it. The extension by weights was continued the whole time, and it was found that the limb became much straighter under it than it had before under a long course of extension without the steaming. This case was the one in which the most marked benefit was observed ;

but in others I have noticed advantage from this method. In another case the leg extended considerably under the continuous steaming, but did not get straight enough for my purpose, so I gave chloroform, and found the adhesions give way with more ease and less damage to the surrounding parts than usual in such cases. This line of practice, viz. continuous extension, and then if sufficient straightening be not obtained, giving chloroform and breaking down adhesions gently should be used in all cases. I am not much in favour of dividing the ham-string tendons, though it has been done in some of the cases in the tables (Cases 9 and 27). With respect to the continuous steaming, the efficacy of the process is believed to depend upon the continuous maceration in the hot, moist atmosphere causing a greater blood-supply to pass to the adhesions and contracted muscles, and thus predisposing them to stretch out under the pressure of the continuous extension. The softening induced in this way may make these adhesions give way under chloroform with less violence than without it. It is possible that continuous poulticing the whole limb, if it could be efficiently done, would have much the same effect.

## II. The Method of Excision adopted.

Nearly all the cases in the tables have been operated on under the carbolic spray, and very much of the success obtained I attribute to the rigid application of all antiseptic details. In all the later cases I have been even more than ordinarily careful about the cleansing of the skin of the joint immediately before the operation. For this purpose I do not trust to soap and carbolic lotion alone, but rub the skin quickly over (especially in cases where the epidermis has become thickened and discoloured) with liquor potassæ. This detaches and dissolves the superficial layer of epidermis, and acts as one of the most efficient skin parasiticides that I know. It should only be very lightly and quickly rubbed on the more delicate skin at the back of the joint, or this may become slightly abraded; and the solution should all be removed immediately after the application by bathing the joint in carbolic lotion.

The straight transverse incision (Esmarch's bandage

round the thigh, used simply as a tourniquet, having been first applied) has been employed in nearly all the cases. The horseshoe incision was used in some of the earlier ones; but it was soon found that the transverse incision was abundantly sufficient for all practical purposes. The exact incision adopted is not perhaps of much importance, except with reference to antiseptic details, and to the method of immobilising the limb adopted after the operation. But it will be found much more difficult to maintain the antiseptic condition of the wound with any other incision than the transverse. Other incisions also involve a much more extensive wound than the transverse, and consequently take longer in healing. Incisions like the H or the horseshoe should be kept for cases where abscesses or the condition of the sinuses absolutely demand it.

The level of the transverse incision should be at the lower part of the patella, and it should be carried as far as the back part of the sides of the condyles. The skin being then lifted from the surface of the patella, this bone is removed by a transverse cut made through the attachments of the quadriceps and retinacula patellæ, and by a second through the ligamentum patellæ.

The *treatment of the patella* has been a much debated point; but in all these cases the patella has been removed. I am very strongly of opinion that this is the most scientific thing to do, but must discuss this later on.

The joint is now forcibly flexed (in which action the softened crucial ligaments generally give way, if the limb has been kept straight for any length of time previously). The head of the tibia is next freed for a very short distance round the margin of the bone, and a thin slice taken off with Butcher's saw, cutting from behind forwards. The depth of this slice must be only sufficient to remove all the articular cartilage, and fairly open the cancellous tissue of the epiphysis. Its depth will vary a little according to the age of the patient, but it may be given, as an average, at a quarter to one third of an inch. The freeing of the fibroid tissue at the back of the tibia from the bone should only be done to a very limited extent—just the amount required for the removal of the bone. If it be done more freely,

there will be risk of bagging of inflammatory material behind the tibia, and the subsequent formation of a popliteal abscess—which always delays the case, and causes a good deal of trouble. It is for this reason that it is recommended that the cut through the bone should be made from behind forwards.

The femur should next be freed from the surrounding fibroid tissue for the necessary extent, and a slice be taken from it. The depth of this cut, as before indicated, may be considerably greater than that of the slice taken from the tibia. The tibial epiphysis is a very shallow plate of bone; that of the femur is much greater.[1]

The practical rule I generally give to myself as regards the depth of the femoral slice is regulated by the depth of the intercondyloid notch. I make a transverse cut just so deep as to remove an exceedingly thin slice of bone above the bottom of this notch.

[1] The depth of these epiphyses, as measured in the adult a little after the age of puberty, in the specimens in the Guy's Museum, was found to be as follows:

Average depth of femoral epiphysis—

As measured in a section of the bone over the inter-condyloid notch, nearly one inch; over the condyles, about one inch and an eighth.

As measured at the sides—the epiphysis still adhering to the bone—about an eighth of an inch greater than these, owing to the concavity of the upper surface of the epiphysis.

Average depth of tibial epiphysis—

As measured in a section of the bone, over the tuberosities, barely half an inch; through the most prominent part of the spine of the tibia, about five eighths of an inch.

As measured at the margins, with the epiphysis still adhering to the bone, in two very strongly developed bones, five eighths of an inch; at the thickest part, six eighths of an inch.

It need scarcely be said that for operative purposes the measurements taken from the section of the bone are much more reliable than those from the margin; and it is manifest that the *smallest* thickness of the epiphysis should be much more taken into account than the greatest. It is clear, therefore, that a considerably thicker slice may be taken from the femur than from the tibia without damaging the growth-lamella. When it is remembered in addition that the facet for the superior tibio-fibular articulation is always on the epiphysis and not on the diaphysis, and that it is not wise to damage this articulation in the operation, the reasons become all the stronger for only taking a very thin slice from the tibia and a thicker one from the femur.

The depth of the epiphysis in children is less than the above in accordance with the age. Corresponding care, therefore, in these young patients should be taken not to remove too much bone.

In all the later cases, the slice has been removed as nearly transversely as possible. The femur or tibia is held vertically by the assistant, and then the saw is carried across it horizontally. In some of the earlier cases I tried various devices with the object of preventing the bones slipping. The tibia and femur were cut obliquely, so that the surfaces corresponded to each other; or the one was cut into a notch and the other into a projecting angle; or the one was cut concave and the other convex to fit into each other. This last plan has been warmly advocated of late by Dr. Fenwick, of Montreal (*vide* 'Ashhurst's International Encyclopædia of Surgery,' vol. iv, p. 536). But all these plans have the disadvantage that they cut more of the epiphysis away in some part than is absolutely necessary; they make also the removal of the bone more difficult, for it is not easy always at the first cut to make the surfaces exactly correspond. The great argument against them is, however, that they are quite unnecessary. Having recognised the necessity of extending the limb as far as possible before operation, I have not found the slightest difficulty in any one of my late excisions in maintaining the limb straight after the operation, when only a plane transverse section of the bones has been made. Of course, much must depend upon the appliances used to fix the limb after the operation, but of that I must speak later on in this paper.

Very much the same criticism may be preferred against the various methods of fixing the bones by pins and sutures after the sections are made. They are unnecessary. They do not do much harm, if the surgeon likes to waste his time in using them, but what little influence they have, seems to me for harm rather than good. Some years ago, my then colleague at the Evelina Hospital for Sick Children, Mr. Morrant Baker, had some steel pins made for fixing the bones. Two of these pins were to be driven rather obliquely from before backwards and upwards through the front surface of the tibial epiphysis into that of the femur. The stems of the pins were to be left projecting either through the incision, or through independent orifices in the skin, so that they could be withdrawn when no longer necessary. I tried these pins in sundry cases; but I found that in all the

young cases, where the cancellous tissue of the epiphysis was loose, spongy, and very vascular the pins caused absorption of the bone round the hole with such rapidity that they become loose in two or three days after the operation, and consequently were of little value in fixing the bones. In adult cases they remained fixed for a longer period, but even here they loosened sooner than might have been expected. These pins were driven in by hand-pressure, and it was manifest that it was difficult in all cases to be certain where they had gone to. In some cases, to get any hold on the bones, they must pass right through the epiphysis and growth lamella of the femur into the spongy tissue of the diaphysis beyond. These pins then add a complication and further risk to the operation, and, being quite unnecessary, should be avoided. I have seen ivory pegs made use of for the same purpose. These were either arranged so that they could be withdrawn, or they were cut short, and left to be imbedded and absorbed. Wire, silk, and gut sutures, for suturing the bones together, I have also tried. Of all these, the verdict must be pronounced :—Unnecessary, adding complications to the operation, causing increased risk, and with no corresponding good result.

*Treatment of sequestra, pulpy cavities, and caseous degenerations of the bone.*—In a very considerable number of cases, on making a section of the bones, it will be found that they are not perfectly healthy, but that they have in them : (*a*) loose sequestra of eburnated or caseous bone lying in cavities lined with granulation-tissue ; or (*b*) patches of caseous bone, not yet separated, but still continuous with the surrounding inflamed bone ; or (*c*) patches in which the osseous tissue has nearly if not quite disappeared, and is replaced by gelatinous or pulpy granulations. If these changes have gone on to any great extent, many surgeons take away another slice of bone, and so on slice after slice until they reach healthy bone. In this way very often the whole epiphysis and growth-lamella of the bone is sacrificed. This interferes with the subsequent growth of the limb, and causes greatly increased shortening from the removal of so much bone. I believe this practice to be altogether bad, especially in children. All such cavities should have their

sequestra removed, or should be gouged out until walls of healthy bone-tissue are reached. A groove may then be cut from the cavity to the surface of the bone, so that when the femur and tibia are applied together, there shall be space for the escape of secretions forming in the cavity. If it be large a small drainage-tube should be inserted; but, as a rule, the groove is sufficient, and no arrangements for drainage will be necessary. This line of practice should be carried out, even where the abscess cavity or caseous bone passes beyond the growth-lamella into the diaphysis; only in such cases the cavity is generally of such large size that a small drainage-tube will be required. The amount of damage done, and the size of the abscess-cavities which may thus be treated with success, is very great. In one case (No. 41) in a child, aged two and a half years, I found a caseous abscess extending from the epiphysis through the growth-lamella half way down the centre of the shaft of the tibia. The epiphysis and growth-lamella were hollowed out by this abscess into a cavity, the walls of which were formed by cartilage lined by a very thin layer of bone. The aspect of this case was so hopeless that I decided immediately that the limb must be amputated; but as I had not obtained the consent of the parents to any such proceeding, I made a drainage aperture, introduced a small elastic tube to the bottom of the cavity, and put it up temporarily as an excision case, pending such time as I could obtain the consent of the parents to the major operation. This consent was refused. To my great astonishment the child did well; the wound healed, and the long drainage-tube was gradually shortened and finally removed. This child has been seen many times since the operation, and it is not the least striking fact about the case that the limb has grown to a degree which I should have thought beforehand quite impossible. On February 25th, 1885, nine years after the excision, the note made by Dr. Pitt is:—"Leg straight, strong, and sound; only 2¼ inches shortening." At the operation only a bare ring of epiphysis and growth-lamella can have been left, and yet it has proved sufficient for the growing capacity of the bone. Such a case as this, which is far from being an isolated example, goes far to show how difficult it

is to be certain before the operation which are the cases likely to do well. This case, therefore, may also be used as an argument against the too close selection of cases for excision. The limb would undoubtedly have been sacrificed if we had been permitted to use our own judgment. A careful selection of cases for excision would undoubtedly produce better results than those in the tables, but it would also sacrifice limbs which might have been saved by the operation.

*Treatment of the thickened pulpy membrane.*—The experience of these cases has led me to the conclusion that the best treatment for redundant pulpy material is to dissect it away to a certain extent, but not to attempt, as many surgeons do, to dissect it all away. In many cases the pulpy material exists in masses an inch or more in thickness. In these it is infiltrating, not the synovial membrane alone, but also the ligaments and periosteum of the bone. Of the bones it principally tends to affect the periosteum of the femur, extending often a considerable distance above the joint. It rarely affects the tibia or fibula to anything like the same extent. This pulpy material, as is well known, infiltrates the normal structures of which the ligaments and periosteum are composed, separating the bundles of fibrous tissue from each other, thus weakening the ligaments in proportion as the fibrous tissue is separated into different fasciculi, separating also the normal blood-vessels of the periosteum from each other, but only rarely producing any fresh growth of blood-vessels—at any rate at all proportionate to the bulk of tubercular material produced. Indeed, many of the normal blood-vessels of the tissue thus infiltrated become obstructed, either by becoming stretched out, or by direct pressure of the growing cells upon them. The parts supplied by these vessels then undergo the various degenerating changes which we recognise as caseous; and hence the patches of cheesy material so frequently found around joints which have undergone old tubercular inflammations. Consequently we have to recognise that, when there is any great formation of tubercular material in the ligaments or periosteum there is no proportionate development of blood-vessels, very few indeed are formed beyond those which were normally contained in the tissue infiltrated. The grey translucent appearance of

tubercular material is due to this cause; it is but rarely pinkish in tint, and when it is so the colour is more commonly due to small extravasations of blood in its interior than to any new growth of blood-vessels.[1] If, therefore, we dissect away all of this infiltrated periosteum, we must necessarily dissect away the principal of the blood-vessels, which in the normal periosteum supplied the bone with blood. In some of my earlier cases I did this, believing that the pulp was a pernicious material which ought to be got rid of at any cost, and hence should be dissected or scraped away to the best extent of our ability. But I found that the cases in which I had done this were more liable to after-troubles, *i. e.* want of union of the bones, either partial or complete, small necroses occurring at the edges of the bones, or subsequent caseous changes in the bone itself, than the others in which I had not been so careful to remove all the pulp. On the other hand, if all the pulp is left, we become subsequently more liable to the formation of caseating abscesses, which have to be opened and scraped out. The best treatment appears to lie in the happy mean. Where the pulpy material infiltrates only the synovial membrane it may be dissected or scraped away as freely as possible. The function of the synovial membrane is at an end after excision, and the more it can be got rid of the better. But where the pulp exists as an infiltration into periosteum, very much more care should be taken of it. I have arrived at the conclusion that it is best to dissect off any very large projecting masses, as, for example, where it is an inch or more in thickness. And where it is of a loose and flocculent character, so that it can easily be broken down or torn off with the finger, it is best to do so. In fact, I make the strength of my finger my test of the degree to which the pulp should be got rid of. And of course, where caseating abscesses occur in the pulpy masses, they should be opened,

[1] The term "pulpy material" or "pulp," so frequently used in this paper, was given to tubercular deposit about joints, &c., by Sir Benjamin Brodie on account of its consistence and greyish colour. It is an exceedingly expressive term, and hence I have preferred to retain it, though it may now be considered as almost definitely proved that it originates from the action of the tubercle bacillus on the tissues.

and the caseating material evacuated, the walls of the cavity being scraped away by the sharp spoon to the greatest degree possible.

In thus advising the only partial removal of the pulp, it is necessary to recollect the changes of which it is capable. Many surgeons treat it as if it were a tumour-formation—the success of the case depending upon its complete removal or destruction. This is, I am sure, a mistake. Pulpy material in its after history is susceptible of three changes:—(1) It may be simply absorbed, just as any inflammatory material disappears by the process of resolution. (2) It may undergo caseation by the process of starvation of a part of itself in the way already described. Or (3) it may organise and become converted into good fibrous tissue. To bring about the changes 1 and 3 should be the object of our surgical treatment. Its conversion into good fibroid tissue is especially desirable, as it helps the consolidation of the part subsequently. The mere *incisions* into the mass of pulp, necessarily made by the operation, do a good deal in producing great changes in it (viz. the draining away of superfluous serum in it, and its consequent contraction, just as incisions into an erysipelatous part helps the cure of it by emptying the blood-vessels and draining away the serum from the tissue). If, in addition, we dissect away superfluous projecting masses, and treat the synovial membrane and caseous abscesses in the way described, I am sure we have put everything in the best train possible for the changes Nos. 1 and 3.

*Arrest of hæmorrhage.*—Excision of the knee is sometimes said to be a very bloody operation. As a fact, no patient need lose more than an ounce or two of blood at the outside. Esmarch's bandage should be applied in Scarpa's triangle before the operation, after a preliminary raising of the limb for two or three minutes. When it is seen that the bone-sections are well adjusted, and the pulpy material cleared away as much as is thought desirable, the Esmarch may be taken off. Up to this time there has been no loss, and the hæmorrhage which now results is generally quite inconsiderable. It can quickly be arrested in the ordinary way. As many of the cut vessels run in the fibrous tissue

close by the bone, *under-running* the vessels with a needle threaded with gut is often the most practicable way of arresting the bleeding.

Sometimes the cancellous tissue of the bone-sections oozes freely. If there is no definite spouting vessel this may be disregarded. It all stops by the pressure applied externally when the limb is put up. If there is a spouting vessel, a little piece of ivory driven lightly upon it stops it. This may be left in. It is, indeed, a good sign to see a considerable number of bleeding points on the removal of the Esmarch, as it argues well for the after-progress of the case. Nothing can be worse than to see pale flabby tissue, the very evascularity of which presages the fate of the limb.

*Treatment of the patella.*—In all the cases in the tables the patella has been removed completely, and this seems to me, on principle, the safest and most successful treatment. It is true that I have not tried any of the more recent methods of conserving the patella. The reasons against doing so seem to me conclusive. I cannot but think that a more extended experience of the various operations for conservation of this bone will prove how far better it is to remove it altogether

The operations in which the patella is saved, are performed—

(α) By opening the joint below the patella, and simply leaving it in the upper flap.

(β) By adding to this operation the removal of the articular cartilage on the posterior surface of the patella, either by scraping it off, or by taking a slice off this surface.

(γ) By opening the joint across the centre of the patella, sawing this bone across, and then wiring it together again after the excision is completed (trans-patellar excision).

(δ) By opening the joint by two vertical incisions, one on either side of the patella, and leaving this bone and the ligamentum patellæ with their attachments intact.

The arguments against all these operations are almost identical. What is the use of the patella after an excision of the knee-joint? Those who advise its retention do so—(1) Upon the ground of a vague conservatism, which believes that the retention of any tissue which normally belongs to the

joint must be an advantage, even when the joint is placed in totally diverse conditions. This argument I have heard used more especially by those who divide the ligamentum patellæ, and simply turn the bone up, retaining it *en masse*. (2) Upon the ground that in the united condition of the parts, when ossification has become complete, it forms a bridge of osseous tissue, extending from femur to tibia, which materially strengthens the union. This is a much stronger argument; but it is to be observed that in excisions in the adult this further strengthening is quite unnecessary. Where bony tissue forms between the sections of the femur and tibia, *i. e.* where no exposed or carious bone remain between them, the union is always abundantly strong. Where exposed or carious bone does remain between them, the retention of the patella will not prevent the necessity for another operation, as a sinus will nearly always remain patent, the discharge and the pain attending which will nearly always lead to amputation—even where the patella has contracted bony adhesions, both above and below, to femur and tibia. In cases where the femur and tibia have united by complete *fibrous* ankylosis, or in a very young case, by very elastic bone, it is possible that this bridge of bone may be of more value, and this might be more especially of avail in very young children to prevent the flexion of the knee-joint, which gradually takes place in some cases, even after a successful excision. It is to be observed here, however, that if strong bony union fails to take place between the femur and tibia, it will also probably fail between the patella and adjoining bones; and that, if only *fibrous* union occurs between the patella and the other two bones, it will probably be quite inefficient to prevent flexion, as the fibroid tissue will gradually stretch out just like the fibrous tissue between the bones. At any rate, it remains with those who advocate the retention of the patella to show by cases that it has a value of this kind. No attempt, as far as I am aware, has ever been made in this direction. Indeed, it would require a very considerable experience of the operation to advance a claim of this kind successfully. If future surgeons should continue to try conservation of the patella, it would be well to take off the cartilage surface of this bone, so as to expose a fresh

osseous surface, and thus facilitate bony union between it and the femur and tibia. (3) Arguments for the retention of the patella are also advanced on the ground that it tends to prevent the backward dislocation of the tibia upon the femur in the period immediately succeeding the operation. It is manifest that those who divide the ligamentum patellæ, and merely turn the patella up, cannot avail themselves of this argument. For this reason trans-patellar excision has been devised. To say nothing of the complication this introduces into the operation, and the consequent lengthening of the time under chloroform, the modification involves distinctly a new risk. There are two bone sections to unite instead of one. It thus combines the risk of the operation of wiring the patella with that of excision of the knee. But the main argument against it is that it is perfectly unnecessary. If the rules which I have laid down for the adjustment of the sections of the femur and tibia be followed, backward displacement *never*, in my experience, takes place. But, even if we were to admit that this displacement were at all a frequent accident in the course of the third and fourth months after the operation of excision, I do not think the retention of the patella *in any way* a method calculated to prevent it. If we examine the cases of pulpy disease of the knee treated by drainage by means of longitudinal incisions on either side of the patella, or those treated by incision and sponging out the interior of the joint with a strong acid (as sulphuric acid)—some of which I have had the opportunity of seeing and following—it will be found that in a certain number backward dislocation takes place, and is a potent factor in preventing the success of the result. Now, in these operations, there is no division of the patella, nor of the ligamentum patellæ, nor of the quadriceps extensor—no solution in fact of the continuity of the quadriceps in its whole length from origin to insertion in the tibia. And yet the displacement occurs. If the quadriceps then be inefficient in preventing backward displacement in cases where it is kept in its full continuity, how much less will it be efficient when it is divided across the centre, as in trans-patellar excision.

This argument applies with equal force against the method

of operating, in which the joint is opened by two longitudinal incisions, one on either side of the joint, and the removal of the diseased surfaces accomplished through these lateral incisions. This operation is an eminently unsatisfactory one. In some few cases, it may be possible (as has been alleged) to turn out the diseased surfaces through the incisions, and thus to see them, and cut away the affected surface. Such a possibility generally implies great laxity of the ligaments, or a very advanced condition of disease. In the great majority of cases the efforts at removal of the bone will have to be confined to scraping or gouging the surfaces, or possibly making a section of the bone by a fine key-hole saw passed from one incision to the other. These proceedings are very unsatisfactory in their nature. No adequate view is obtained of the surfaces of the bones. Remembering how often these are the seat of pulpy mischief, or present patches of caseous osteitis, or even sequestra lodged in granulating cavities, it will be seen how important it is to obtain a full view of the bone after the section is made. To leave bone behind in a diseased condition is to ensure the failure of the operation. One of the very strong arguments for excision is the frequency with which such diseased conditions of the bone exist, and the difficulty of distinguishing them beforehand from the more simple cases. To adopt then a method of operating which renders the recognition and treatment of these diseased conditions very difficult, and in many cases impossible, is simply to do away with the main utility of the operation, and to invite failure. Certainly no supposed advantage obtained by the retention of the patella and attachments of the quadriceps is in any way compensatory to the great disadvantages here shown to exist.

To sum up, the main object of the retention of the patella is:—(1) To prevent backward dislocation—which, so far as my experience goes, never occurs after the ordinary operation. (2) To prevent flexion after the operation. There is no evidence of its possessing any value in this direction, and even if a series of limbs remaining straight some years after this operation were recorded, it would not be a sufficient proof, without comparison with an equal number of cases of

patients about the same age in which the patella was removed, as the flexion is an unusual result after the common operation, only having occurred to any important extent in about 13 out of 130 cases. To form a basis for such comparison is one object I have set before myself in this paper.

*Closure of the wound and drainage.*—Before the final closure of the wound, I think it wise (especially in the pulpy cases) to syringe it out thoroughly with warm solution of perchloride of mercury (1 to 1000). This, in my experience, is better than the carbolic solution, and it seems to me to have some special influence for good on the pulpy material.

I usually close the wound with a quill suture, but only just in front, leaving open the sides for drainage. I use no drainage material, such as horse-hair or tubing, unless there is a distinct cavity which it is necessary to keep open, finding that the lateral parts of the wound, if left unsutured, are quite sufficient for this purpose. The quill suture was adopted so as to evert the skin, and thus prevent any tendency to its dipping down between the bones, which happened in one or two of the earlier cases.

*Mode of putting up the limb.*—Many of the earlier cases were put up in very imperfect apparatus, and hence the number of comparative failures in these cases. For example, the first case, and two or three subsequent ones, were put up in the old box-splint at that time in vogue. Nothing could have been worse mechanically. Two or three of the succeeding cases were put up in a simple straight back-splint, which was taken off and changed nearly every time the wound was dressed. The disturbance of the wound which such a proceeding causes is distressing to think of. But the experience of these cases taught me what an excision case would bear, and what it requires to become successful, and hence we gradually elaborated the present splint. This consists of two *shallow* troughs of sheet iron tinned, made so as to fit the shape of the leg and the thigh. The foot-piece works in a slot in the usual way, so as to allow for elongation or shortening of the leg-piece. The two troughs are connected at the back by two strong bars of iron made convex transversely, so as to avoid cutting into the skin of the popliteal space. Of these bars one is connected to the leg-trough, the

other to the thigh-trough, and they move upon each other by a slot, and are fastened together by screws and nuts.[1] This arrangement permits the interval between the troughs (which corresponds to the excision wound) to be lengthened or shortened according to the case. The interval should never be less, even in the youngest children, than four inches. In the adult six to seven inches is the common one. The splint has also a small pulley at the bottom beyond the foot-piece.

The splint is not padded, but is lined with a layer of lint soaked in a hot mixture of yellow wax and carbolic oil (3 lbs. to 20 oz.) melted together. The only object of this is to prevent the metal coming directly into contact with the skin. The waxed lint forms a watertight material, into which no discharges soak, so that the risk of decomposition is reduced to a minimum.

The wound having been closed and the antiseptic dressings applied, mostly at the front and at the sides, and only very little at the back, so that the popliteal space may rest as directly as possible on the bar, the limb is placed on the splint, and a pad put below the heel (over the tendo Achillis) so as to keep the leg straight. The limb is then fixed in the splint. In doing this the fixing of the thigh is the most important thing to attend to. I usually fix first the thigh-trough to the limb by strapping rather loosely applied (to allow for a little swelling subsequently). Next, I carry a broad soft flannel bandage over it, and figure of 8 round the abdomen, padding the sacrum and crests of the ilium by a layer of Gamgee tissue. Then over the whole I put on plaster of Paris bandages, strengthening them by strips of the same bandages opposite the hip-joint. The object of this is to make the thigh-trough of the splint move in one piece with the body of the patient. It is of especial importance in children, who cannot understand the importance of lying still, and in whom the thigh is very short. It is of less importance in the adult, where the length of the thigh enables the splint to obtain a good grasp of it.

[1] These bars should be made very strong and rigid. The instrument makers generally make them too flexible and yielding. Thus they do not entirely immobilise the joint, and sometimes cause the failure of the operation.

But it has proved so useful in my hands that I now apply it to all excision cases. If it be not used (and it has only been used in the later cases in the tables), it will be found that in a certain proportion of the cases the thigh gradually gets loose, and in the movements of the body in the bed becomes drawn up in the splint, so that the foot gets separated from the foot-piece. This movement brings the incision close under the bandage fixing the thigh. As the knee is slightly larger than the circumference of the thigh immediately above it, the knee gets wedged in under the bandage, producing œdema of the part operated on, and thus retarding healing. It also endangers the safety of the antiseptic treatment. If the leg be very firmly fixed in its trough the femur gets drawn away from the tibia to a certain extent, thus producing a greater interval between the bones than is advisable, and thus tending to delay the period of ossification, and even bringing about in some cases want of union. This loosening of the thigh-piece and alteration of the position of the incision used to be a great source of trouble to me, but now it is quite obviated by the method advocated. It enables also the bandages about the thigh to be much more loosely applied, thus favouring union by the avoidance of any passive congestion.

The fixing of the leg is a much more simple matter. It should simply lie on the leg-trough, fixed as loosely as possible by turns of bandage. I commonly employ a waxed bandage, *i. e.* a calico bandage soaked in the same mixture as that used for the lint on the splint. This is used simply to make it water-tight, so that the spray and discharges may not soak into it and keep it constantly moist. But it is not of much importance. An ordinary bandage, or a plaster bandage, painted over when dry with the wax mixture answers equally well. I paint the lower part of the plaster of the thigh-piece with the same mixture, for the same reason.

The knee should be fixed to the bar between the troughs by an ordinary *calico* bandage (which does not stretch and become loose, as some other materials do). This should be done directly the limb is laid on the splint to avoid the risk of any subsequent shifting of the bones in the after

manipulations of the limb. I always begin by (and lay a considerable stress on) bandaging the upper part of the interval first, so as to secure the thigh before the leg. And I always bandage *from without inwards* (contrary to the usual rule). This is because there is always considerable tendency, as the patient comes round from the anæsthetic for the thigh to rotate outwards. In bandaging from without inwards every turn of the bandage over the femur, as it is drawn tight, tends to counteract this tendency, whereas in bandaging in the opposite direction each turn of the bandage tends to rotate the thigh more outwards. This rule I observe throughout all the subsequent dressings. The disastrous effect of allowing the thigh at this stage to rotate outwards, while the leg remains fixed in its right position, is seen in the subsequent progress of the case. If it is allowed, union takes place between the bones with the femur more rotated outwards than the tibia. This *looks* all right when the limb is lying on the splint; but when the patient begins to walk, the femur naturally restores itself at the hip-joint to the right degree of parallelism with its fellow, and thus rotates the leg, now ankylosed to it, inwards. The result is a very awkward and unsightly turning inwards of the foot, which may be prevented by a little care in the direction indicated. If the surgeon sees, a day or two after the limb is put up, that there appears to be some eversion of the whole limb, as shown by the foot rotating outwards, he need not be disturbed. The rotation has probably taken place from the hip, and the accuracy of the fixing is shown by the whole limb (leg and splint) rotating with it.

A Salter's swing cradle is used for suspending the limb. Below the wheels running on the longitudinal bar, I add another small pulley placed on a plane at right angles with them, and corresponding to the pulley already mentioned on the end of the splint.[1] The lower end of the splint is

[1] A diagram of this arrangement, as employed about 1877, was placed in the 'Guy's Hospital Reports' for that year (vol. xxii, series iii, p. 502). It does not represent the rotatory pulleys now added to the splint and cradle, nor does it represent the mode of fixing the splint to the pelvis, alluded to in the text; but it sufficiently indicates the method to render it unnecessary to present any modification of the drawing on the present occasion.

supported by a cord passing round the two pulleys. This permits free rotation of the limb when the patient turns from one side to the other, and is a great comfort to him, as it does not necessitate his remaining absolutely in one position in bed. It also saves the risk of bed-sore, &c.

*Arrangement of the bed and cradle to permit subsequent dressings.*—Our hard mattresses for adults are commonly divided into three pieces. The lower piece is taken away and replaced by the cradle. A pillow is placed alongside of the cradle for the other limb to rest on. Both cradle and mattresses are placed on boards to prevent any sinking down (the usual sacking or spring beds are not advisable). Formerly I used to have a quarter water-pillow or bed placed beneath the buttocks. With our improved means of putting up the limb this has now become unnecessary. The excision wound in the gap in the splint, therefore, lies below the mattress (*i. e.* nearer the foot of the bed), in a space in which the hands can freely work while dressing, without disturbing the limb in the splint in the slightest degree. The dressings thus become almost entirely painless to the patient (with the exception perhaps of the first, when the clotting of the fresh blood causes the gauze to stick a little to the skin).

Carbolic gauze oiled with carbolic oil (1—16) to prevent sticking to the skin, and iodoform, are used for the dressings with the spray. The first dressing is done generally forty-eight hours after the operation; the quill suture is then removed, and any wire sutures (used to bring the skin together above the quill) taken away in the subsequent dressing. All risk of inversion of skin between the bones is then at an end. The dressings are continued at gradually increasing intervals in accordance with the amount of discharge, and progress of the wound towards healing. Of late I have added to the above a certain amount of sal-alembroth gauze (about eight thicknesses), and have increased the intervals between each dressing very considerably after the first two (removal of the sutures),—with advantage, I think, to the patient.

*Treatment of subsequent abscesses and pulpy sinuses.*—

Whatever be the method adopted of dealing with pulpy material, abscesses will form and sinuses remain in a certain proportion of excision cases. The abscesses are most frequently due to breaking down of masses of pulp, or to caseating cavities in it, not perceived at the time of the operation. It may be argued that these would have been avoided by a freer treatment of the pulp at the time of the operation, as by dissecting it all away. This may be true; but, as I have already shown, dissecting the pulp away not infrequently introduces a new element of danger in damaging the blood-supply to the bones, and hence we get another cause of abscess in these cases. Taking it all together, I think the breaking down of some superfluous pulp left behind at the time of the operation is a far less evil than the damaged blood-supply to the bones. The great thing after excision is to get the bones to unite satisfactorily. If this takes places, it is in but very few cases that remaining pulpy tissue may not be dealt with satisfactorily. The rule with me is to open acute abscesses wherever they appear, and if in their interior I find caseating material to break it down and bring it away with the finger. In more chronic abscesses to open them freely, and scrape the interior of the cavity with the sharp spoon. With sinuses to incise them freely, scrape them out with the spoon, and often to sponge out the interior with nitric acid on a wad of cotton-wool. I have had to repeat this process sometimes seven or eight times in a single case, where the tendency to pulpy infiltration was great. It is of course always done under chloroform. On the other hand, if I find three or four months after the excision the sinuses running down between the bones, with a want of union between them, and very small power of developing sound connective-tissue, I regard it as an infinitely worse case than if they are connected simply with masses of pulp. In many of my earlier cases I struggled long and hard to save these limbs, by scraping the bones, by swabbing them with strong nitric acid, and other means. I found very few cases indeed repay treatment. These limbs mostly have to be amputated. At the present time, if the want of union be slight, I may once or twice think it worth treating the sinuses in the same way. But if it be

at all extensive, I think it is only increasing the danger to the patient of some subsequent lardaceous or general tubercular trouble, and consequently advise the patient to sacrifice the limb.

*Medicinal treatment of the patient.*—Many of the cases have been treated after the operation with a course of perchloride of mercury (dose $\frac{1}{12}$ grain, three times a day, taken directly after food). This has been used not only in those in which the trouble is supposed to be of syphilitic origin, but also in those where it is believed to be purely scrofulous. It is always difficult to assign exactly the degree of influence to be ascribed to one particular remedy in any complex treatment; but I think I have seen great benefit from its use in both classes of cases. The benefit received has been sufficiently marked to make me think I ought to add this note to the paper.

Other constitutional remedies have of course been used. Cod-liver oil, iron, quinine, and arsenic have been the principal. Their use calls for no comment.

*Comparison of Results in the Earlier Cases operated with the Later.*

If the results of the first twelve cases in the following tables be compared with the last twelve, a striking increase in the successful results is noticeable. The causes of this have been before referred to.

If these results are placed in tabular form it will be seen that:—

| Of the first dozen— | Of the last dozen— |
|---|---|
| 2 were entirely satisfactory. | 7 were entirely satisfactory. |
| 2 were satisfactory. | 1 was satisfactory. |
| 3 were flexed. | 1 was very slightly flexed. |
| 3 were amputated. | 2 were amputated. |
| 2 died. | 1 died. |
| 12 | 12 |

The case here put down as "slightly flexed" may from the after-history be regarded as "entirely satisfactory."

Still more strikingly do the improved results come out if the first score of cases be compared with the last twenty.

It is worth while to give these results in detail as taken from the tables. It will thus be seen what is the signification which is attached to the terms "satisfactory" and "entirely satisfactory" in this and the preceding table.

*Comparison of Results in the First Twenty Cases operated on with the Last Twenty.*

THE FIRST TWENTY.

Case
1.—Well. Ankylosed. Backward dislocation. Satisfactory.
2.—Well. Flexed.
3.—Well. Flexed.
4.—Death. Gangrene.
5.—Well. Amputation.
6.—Well. Flexed. Wedge-shaped piece removed, and leg amputated 9 years afterwards.
7.—Well. Entirely satisfactory.
8.—Well. Amputation.
9.—Well. Entirely satisfactory.
10.—Well. Amputation.
11.—Death from tubercular meningitis.
12.—Well. Satisfactory.
13.—Well. Entirely satisfactory.
14.—Well. Entirely satisfactory.
15.—Well. Amputation.
16.—Well. Entirely satisfactory.
17.—Well. Entirely satisfactory.
18.—Well. Amputation.
19.—Well. Satisfactory.
20.—Well. Entirely satisfactory.

THE LAST TWENTY.

Case
129.—Well. Very slightly flexed.
128.—Well for 1½ years. Entirely satisfactory, but died at home 1½ years after from some zymotic affection. Case included as satisfactory.
127.—Well. Entirely satisfactory.
126.—Well. Entirely satisfactory.
125.—Well. Entirely satisfactory.
124.—Well. Entirely satisfactory.
123.— ? Amputation.
122.—Well. Entirely satisfactory.
121.—Well. Entirely satisfactory.
120.—Well. Amputation.

119.—Death. General tuberculosis.
118.—Well. Entirely satisfactory.
117.—Well. Good on discharge. After-history unknown.
116.—Well. Good on discharge. After-history unknown.
115.—Well. Good on discharge. After-history unknown.
114. } Well. Two excisions in one patient. Both did well.
113. }
112.—Much pulp. After-history unknown.
111.—Well. Very slightly flexed.
110.—Well. Entirely satisfactory.

To sum up the results in these cases :

Of the first twenty patients—
7 were entirely satisfactory.
3 were satisfactory.
3 were flexed.
5 were amputated.
2 died.

Of the last twenty patients—
11 were entirely satisfactory.
4 were satisfactory on discharge, but after-history unknown.
2 were very slightly flexed. These may practically be included as entirely satisfactory as far as the patient is concerned.
2 were amputated.
1 died from general tuberculosis.

These are nearly all of them results obtained many years after the actual date of the operation.

# TABLE OF CASES.

| No. | Initials, sex, and age. | Disease. | Duration of disease before excision. | Previous treatment. | Admission and discharge. | Duration of stay in hospital. | Operations performed. |
|---|---|---|---|---|---|---|---|
| 1 | E. W., F., 21 | Old pulpy disease, with extreme backward dislocation. Left knee | 10 years | Various splints, in hospital. Medical. Always walked with crutches | A. Feb. 7, 1872<br>D. Feb. 15, 1873 | 373 days<br>After operation 352 days | Excision Feb. 28, 1872<br>Necrosed end of femur removed in June, 1872<br>Sinuses incised Sept. 16, 1872, and on Nov. 8, 1872 |
| 2 | F. T., M., 10 | Pulpy, right knee, with backward dislocation | 6 years | Splints. Medical, &c. | A. Sept. 24, 1872<br>D. June 15, 1873 | 265 days<br>After operation 228 days | Excision Nov. 1, 1872<br>Abscess opened on right side Jan. 22, 1873 |
| 3 | R. S., M., 8 | Pulpy, left knee | 4 years | In St. George's and Great Ormond Street Hospital twice | A. June 26, 1871<br>D. Feb. 14, 1872<br>Re-admitted Oct. 2, 1872<br>Re-discharged Oct. 4, 1872 | 233 days<br>After operation 222 days | Excision July 7, 1871 |
| 4 | R. T., F., 10 | Pulpy disease. Left knee | 6 years | Rest. Medical | A. April 24, 1873<br>Death June 4, 1873 | 42 days<br>After operation 26 days | Excision May 10, 1873 |

| State of joint at time of excision. | State on discharge. | After history. | Remarks. |
|---|---|---|---|
| Total destruction of joint. Extreme backward dislocation. Bone-sections healthy. Want of development of left leg and foot, left tibia being ¾ of an inch shorter than right. Foot the same, the whole limb being proportionately short and ill-developed. 1 inch of bone cut from femur, ½ inch from tibia | Bony ankylosis, but tibia united to posterior surface of femur on account of slipping of the bones after operation. Walked well with stick. Total shortening 4 inches | Seen many times since her discharge. On the last occasion had walked 4 miles to the hospital without inconvenience, but wears a patten on the affected side | The great shortening of the limb was due in this case partly to previous atrophy of the limb, partly to accidental slipping of the bones 48 hours after the operation, when the nurse was changing the sheet. Old box-splint employed. Limb not slung. Not enough bone cut away to allow parts to lie easily in position without straining. |
| Complete destruction of joint from pulpy disorganisation. Backward dislocation | Wound healed. Fibrous ankylosis at a slight angle. No shortening | Last seen in April, 1877. Apprenticed to a printer, where he smashed a finger, and came in to have it amputated. Firm, apparently bony ankylosis, but at a considerable angle | For subsequent osteotomy. |
| Pulpy disease with sinuses and backward dislocation | Walks well without pain. Wounds entirely closed | Seen 1½ years after discharge. Continued to walk well without any protection to the leg, but the angle had increased considerably. Growth of limb normal | Very considerable hæmorrhage in night following operation. Length of stay in hospital partly due to this cause, partly to imperfect appliances. |
| Suppuration of joint. Ligaments all vascularised and softened. Bone-sections healthy | Death. P.M.—Gangrene of opposite limb up to knee from too tight bandaging. General pyæmia. Excision-wound nearly united | Amputation was advised, but parents refused consent | Child with excessively feeble capillary circulation. Opposite limb bandaged, and one pound weight applied by dresser to prevent the child knocking the excised limb with it. Child never complained, but the circulation failed. |

| No. | Initials, sex, and age. | Disease. | Duration of disease before excision. | Previous treatment. | Admission and discharge. | Duration of stay in hospital. | Operations performed. |
|---|---|---|---|---|---|---|---|
| 5 | E. M., F., 21 | Pulpy, right knee | 3 years | In Colchester Hospital twice for some months each time | A. Sept. 5, 1873<br>D. April 3, 1874 | 7 months<br>After operation 157 days | Excision Oct. 28, 1873<br>Amputation Jan. 14, 1874 |
| 6 | E. B., F., 3 | Pulpy disease with backward dislocation | 20 mos. | In Charity Ward under Mr. Forster on July 30, 1873. Limb straightened under chloroform. Splint | A. Sept. 29, 1873<br>D. May 29, 1874 | 8 months<br>After operation 8 months | Excision Sept. 30, 1873<br>Mar. 3, 1882, amputation through thigh |
| 7 | T. G., M., 38 | Pulpy, left knee | 6 years | In Ramsgate Infirmary 7 weeks, but has continued about upon the leg with crutches more or less to the present time | A. Nov. 11, 1873<br>D. May 7, 1874 | 6 months<br>After operation 5½ mos. | Excision Nov. 21, 1873 |
| 8 | J J., M., 2 | General pulpy disease of elbow and knee. Probably congenital syphilis. Child suffered from universal eczema | Elbow 1½ yrs. Knee 10 mos. | Medical and splints | A. Feb. 8, 1873<br>D. (?) | ? | Excision Aug. 30, 1873<br>Amputation about 1 year after |

| State of joint at time of excision. | State on discharge. | After history. | Remarks. |
|---|---|---|---|
| Complete disorganisation and backward dislocation. Cartilages all gone. Bone surfaces lined with granulation. Joint full of pus | Stump with one sinus remaining open, which communicated with a large abscess sac, reaching up the thigh; this existed previous to amputation | Sinuses entirely closed 3 or 4 months after her discharge from hospital, and she was able to get about fairly actively upon an artificial leg, but she ultimately died of phthisis about 2 years after her discharge | No evidences of lung disease at time of excision, but a severe attack of hæmoptysis came on 3 weeks after operation. On this account, and as wound was in a very hæmorrhagic state, amputation was recommended a day or two afterwards. She absolutely refused her consent, however, for nearly two months more, during which time a large abscess formed in thigh and extended up to trochanter. When almost *in articulo mortis* she gave her consent. Amputation in middle of thigh through abscess sac. Rallied after operation, and went out as before described. Absolutely no union between the excised ends of the bones. |
| Backward dislocation. Ligaments softened. Synovial membrane pulpy | Wound healed. Firm fibrous union, but with slight flexion and rotation | Seen in spring of 1877. Walking well upon the leg, but flexion and rotation has considerably increased, and leg appears considerably distorted. Growth of bone normal. For osteotomy when patient is older.<br>Dec. 30, 1879: Re-admitted. Leg flexed at right angle and firmly united to femur, but rotated inwards. Growth of bone in popliteal space. Wedge-shaped piece of bone removed from head of tibia Jan. 17, 1879. Went out May 31, 1879, with bones straight and united, but tibia still rotated much inwards.<br>Feb. 24, 1882: Tibia bent at obtuse angle with femur, and rotated inwards. There is $4\frac{1}{2}''$ shortening; muscles much atrophied | Mar. 3, 1882: Amputated through lower third of thigh, ant. post. flap.<br>April 6, 1882: Went home, stump all healed, but small spot at outer end of wound. |
| Crucial ligaments gone. Articular cartilage eroded and flaking off. External tuberosity of tibia with caseous patch in it | Bony ankylosis. Limb perfectly straight. A slight superficial sore remaining, but could walk well | Was seen by a patient plying his trade as a boatman three years after the operation | — |
| General pulpy disease. Osseous nucleus of epiphysis of femur, and tibia reduced to consistence of red currant jelly and turned out with finger. Many sinuses | Stump healed | — | Lardaceous disease showed itself in the liver about 9 months after excision. On account of its progressive increase amputation was resorted to one year after excision. Liver returned to its normal size again. The eczema disappeared after excision. |

| No. | Initials, sex, and age. | Disease. | Duration of disease before excision. | Previous treatment. | Admission and discharge. | Duration of stay in hospital. | Operations performed. |
|---|---|---|---|---|---|---|---|
| 9 | M. S., F., $10\frac{3}{4}$ | Pulpy, right leg | 3 years | Rest. Tendons divided. Had been in hospital once or twice | A. Nov. 6, 1874<br>D. May 6, 1876 | 18 months<br>After operation 15 months | Excision Feb. 13, 1875 |
| 10 | (?) F., 10 | Pulpy, (?) side | 2 years | Rest. Medical | (?) | About 5 months | Excision. Amputation through thigh 1 month after |
| 11 | E. H., F., 21 | Pulpy, left knee | 6 years | Rest. Blistering. Medical. In hospital (Guy's) 13 weeks | A. Feb. 3, 1874<br>Death March 25, 1874 | 7 weeks<br>After operation 40 days | Excision Feb. 13, 1874 |
| 12 | A. B., F., 12 | Genu-varum, with great looseness of ligaments, &c. Right knee | 2 years | Rest. Strapping. Medical | A. Apr. 22, 1874<br>D. Sept. 9, 1874 | 140 days<br>After operation 132 days | Excision May 1, 1874 |
| 13 | E. C., F., 22 | Old pulpy disease, with caseous necrosis in joint | From child-hood | Twice in St. Thomas's Hospital for 4 and $3\frac{1}{2}$ months respectively. At various places elsewhere | A. Feb. 19, 1874<br>D. July 15, 1874 | 149 days<br>After-operation 138 days | Excision on March 2, 1874 |

| State of joint at time of excision. | State on discharge. | After history. | Remarks. |
|---|---|---|---|
| Joint structure destroyed. Some backward dislocation. Adhesion between one condyle and tuberosity. Localised collection of pus between other condyle and tuberosity | Sinuses closed, but with some tendency to break down about end of wound when rubbed. Able to walk on leg. ½″ shortening | Apr. 12, 1879: wears back splint; leg strong and straight. Jan. 1, 1885: leg straight, sound, and strong; health good; very little shortening | Became subsequently a nurse, and is now doing duty in this capacity. |
| Extensive disorganisation of joint | Stump healed | Unknown | A case at the Evelina Hospital, where the report was unfortunately lost. |
| Joint structures entirely destroyed. Articular surfaces of femur and tibia coated with granulation. Granulation replacing cartilage on patella formed into a kind of smooth membrane. Much caseous pulpy stuff | Tubercular meningitis came on 3 weeks after operation, and proved fatal in about 3 weeks more. (Headache first began on March 4th, 1874) | At P.M. the adhesion was found so firm that it only broke down when the whole weight of the body was thrown on that leg | — |
| Great development of the external condyle, with total arrest of growth in the internal. Great looseness of the ligaments | Walking well. Shortening scarcely one inch | Seen July, 1877. No increase of shortening. Limb perfectly straight and firmly ankylosed. Has had some threatenings of chest trouble | Case reported in 'Guy's Hospital Reports,' vol. xx, p. 531. |
| Strong fibrous ankylosis between patella and femur. Cartilages all gone. Articular surfaces of bone considerably altered in shape. Internal condyle of femur presented a cavity open to the remains of the joint-cavity, containing a caseous sequestrum; surrounding bone caseous | Firm ankylosis. Patient walking well. Shortening ½ inch | Seen many times since operation. Patient continues perfectly well and able to do her household work. Jan. 1, 1885: leg straight, sound, and strong; some shortening; health good | — |

| No. | Initials, sex, and age. | Disease. | Duration of disease before excision. | Previous treatment. | Admission and discharge. | Duration of stay in hospital. | Operations performed. |
|---|---|---|---|---|---|---|---|
| 14 | S.J.H., F., 19 | Pulpy, right knee | 5 yrs., much worse 3 mos. | Rest, with splints. Medical | A. July 16, 1874<br>D. Dec. 19, 1874 | 156 days<br>After operation 106 days | Excision Sept. 4, 1874 |
| 15 | K. T., F., 6 | Pulpy disorganisation of right knee-joint | 1 year | Knee-joint had been previously incised on July 6, 1874. Stinking discharge and much formation of pulpy material followed | A. May 6, 1874<br>D. May 24, 1874<br>Re-admitted June 29, 1874<br>Re-discharged Feb. 11, 1875 | 1st, 18 days<br>2nd, 227 days | Excision Sept. 29, 1874<br>Amputation Dec. 8, 1874 |
| 16 | M. J., M., 30 | Pulpy, left knee | 2 mos. | Rest, with splints. Medical. Joint tapped; effusion quickly returned | A. Sept. 30, 1874<br>D. July 22, 1875 | 9¾ mos.<br>After operation 8 mos. | Excision Nov. 14, 1875 |
| 17 | J. H., M., 22 | Gonorrhœal rheumatism, with pulpy, left knee | 3½ mos. | Splints. Strapped with Ung. hyd. Medical | A. Aug. 15, 1874<br>D. April 8, 1875 | 8 months<br>After operation 4½ months | Excision Nov. 20, 1874 |
| 18 | H. S., M., 24 | Pulpy, with hydrarthrosis, left knee | 3 years | In hospital from Oct. 28 to Nov. 12; knee-joint tapped, 2¾ oz. of fluid withdrawn. Medical, and splints | A. Jan. 7, 1875<br>D. Sept. 29, 1875 | 8¾ months<br>After operation 8¾ months | Excision Jan. 12, 1875<br>Amputation Sept. 27, 1876 |

| State of joint at time of excision. | State on discharge. | After history. | Remarks. |
|---|---|---|---|
| Joint structures entirely gone. A small bony ankylosis taking place superficially at one point. Caseous bone externally | Able to walk about ward. Limb firmly ankylosed | Jan. 1, 1885: leg straight, strong, and sound | Patient of weak intellect. |
| Bone sections healthy. Joint surrounded by an enormous mass of pulpy material, caseating and suppurating in various directions | Thigh stump healed | Seen in spring of 1877. Quite well | Much union took place after the excision, but two sinuses remained. Albuminuria set in at end of November. Thigh amputated on Dec. 8th. Rapid improvement. Albumen disappeared by end of month. Stump quite healed by middle of January. |
| Synovial membrane swollen and pulpy, encroaching on outer side on the cartilage. Bone sections healthy | Excision quite healed. Firm ankylosis. Walking well upon it. $\frac{3}{4}$ inch shortening | Seen in summer of 1877. Quite well, and able to do ordinary labourer's work. Walks with scarcely perceptible limp | — |
| Synovial membrane much thickened. Pulpy and vascular fringes projecting into joint. Crucial ligaments present but softened. Erosion of cartilage on inner side. Bone sections healthy | Limb firmly ankylosed. Walks well | Wrote on Oct. 18, 1875, that he was able to do twelve hours' work in the day without the slightest pain or weakness in the leg | — |
| Synovial membrane swollen and gelatinous; considerable erosion of cartilage from both condyles. Crucial ligaments softened. Bone sections healthy | Knee ankylosed. Two or three sinuses remaining open, causing a considerable amount of foul discharge, which gave rise to much unpleasantness | Went to Germany for change of air. Had the leg amputated on his return, Sept. 27, 1876, as the change had not proved beneficial. | — |

| No. | Initials, sex, and age. | Disease. | Duration of disease before excision. | Previous treatment. | Admission and discharge. | Duration of stay in hospital. | Operations performed. |
|---|---|---|---|---|---|---|---|
| 19 | E. T., F., 21 | General osteo-arthritis | 2 years | Principally medical and with splints. Under Dr. Habershon. Right knee straightened under chloroform | A. Jan. 19, 1875<br>D. Aug. 3, 1875 | 7½ months<br>After operation 6 months | Excision of left knee on Feb. 3, 1875 |
| 20 | A.L.A., F., 18 | Complete pulpy disorganisation, right knee | 1 year | Rest, with splints | A. Nov. 24, 1874<br>D. May 3, 1875 | 5¼ months<br>After operation 5 months | Excision Dec. 4, 1874 |
| 21 | E. H., F., 21 | Pyæmic disorganisation. Left knee | 4½ mos. | Rest. | A. Mar. 17, 1875<br>D. Aug. 11, 1875 | 5 months<br>After operation 4½ months | Excision March 30, 1875 |
| 22 | M. D., F., 27 | Pulpy, right knee | 5 years | Various. | A. May 20, 1875<br>D. Oct. 23, 1875 | 5 months<br>After operation 4½ months | Excision June 24, 1875 |
| 23 | E. H., F., 4 | Pulpy disorganisation of knee-joint with open sinuses extending up thigh. Strumous ophthalmia | 2¼ years; abscesses formed 1½ years ago | Rest. Incision of abscesses, &c. Splints. Extension | A. Sept. 9, 1872<br>D. Mar. 17, 1873<br>Re-admitted June 23, 1873<br>Re-discharged June 7, 1874 | 6 months<br>11½ months<br>After operation 9 months | Excision Sept. 16, 1873<br>For closing sinuses on March 25, 1874. Firm union (apparently bony) then stated to exist |

| State of joint at time of excision. | State on discharge. | After history. | Remarks. |
|---|---|---|---|
| Joint structures almost entirely destroyed. Crucial ligaments softened. Patella ankylosed on to femur, so that it could not be separated. Section cut through the conjoined bones | Ankylosis, but on account of the condition of the other joints could not use her leg | Jan. 1, 1885: leg sound, straight, and strong; about 1″ shortening | Aug., 1875: Improved. Out of pain, and could lift up the leg. |
| Synovial membrane thickened and gelatinous. Crucial ligaments destroyed. Articular and inter-articular cartilages destroyed. Caseating bone in centre of tibial epiphysis | Knee ankylosed. No sinuses. Can walk well | Seen several times. Can use leg perfectly, so that it is scarcely possible to tell that the joint has been excised. Scarcely any shortening | — |
| Patella cartilage eroded. Articular cartilage on internal tibial tuberosity entirely eroded. External condyle and tuberosity scarcely affected. Crucial ligaments completely softened | Entirely healed. Walks well | Heard of three or four times since discharge. Able to use her leg and perform her household duties perfectly well | — |
| Bone sections caseous; chiselled out into a groove. Cartilages peeling. External ligaments stretched and loosened | No sinuses. Walking | Seen in summer of 1877. Getting about fairly well, but being a weakly phthisical woman, with a great tendency to disease in her other joints, she is not able to be quite so active as some of the cases. 1½″ shortening<br>1879: Getting about actively considering state of health<br>Jan. 1, 1885: Limb straight, strong and sound; health good | — |
| Patellar cartilage deeply eroded. Ext. condyle and tibial tuberosities bare and covered with gelatinous granulation. Bone sections fairly healthy. Femoral sinuses not much interfered with | Able to walk and play about ward. Leg quite straight. Sinuses closed | Heard of five or six months after discharge. Continued in good health<br>Jan. 1, 1885: Leg straight and sound. Patient says half an inch shortening. No movement between bones, but a little weakness after a long walk | — |

| No. | Initials, sex, and age. | Disease. | Duration of disease before excision. | Previous treatment. | Admission and discharge. | Duration of stay in hospital. | Operations performed. |
|---|---|---|---|---|---|---|---|
| 24 | J. L., F., 10 | Pulpy, left knee; well-marked evidence of congenital syphilis | 4 years | Three times in hospital, rest, with splints, hamstring tendons divided for contraction | A. Dec., 1874<br>D. July 20, 1875 | 7 months<br>After excision 5¾ months | Excision Jan. 30, 1875 |
| 25 | W. K., M., 7 | Pulpy, right hip and left knee | Of knee 1 year | Rest. Splints. Medical. Incision | A. Jan. 22, 1873<br>D. Feb. 18, 1874<br>Re-admitted April 15, 1874<br>Re-discharged Feb. 16, 1876 | 13 months<br>22 months<br>After excision 10 months | Excision of right hip Mar. 25, 1873. Amputation Jan. 2, 1874. Incision of knee May 5, 1874. Excision April 27, 1875 |
| 26 | F. M., F., 14 | Pulpy disorganisation with suppuration of joint | 3½ mos. | Rest. Ice. Medical. Joint tapped | A. Mar. 17, 1875<br>D. May 7, 1875<br>Re-admitted May 29, 1875<br>Re-discharged Feb. 16, 1876 | 1¾ months<br>9 months<br>After operation 7½ mos. | Excision June 25, 1875<br>Amputation Nov. 10, 1875 |
| 27 | S. C., M., 20 | Probably old pulpy disease | 3 years | Rest. Abscesses opened. Twice in provincial hospital for 8 months | A. June 9, 1875<br>D. Mar. 2, 1876 | 9 months<br>After excision 8¼ months | Division of hamstring tendons June 16, 1875. Excision June 29, 1876. Sinuses incised |

| State of joint at time of excision. | State on discharge. | After history. | Remarks. |
|---|---|---|---|
| Patella eroded, fibro-cartilages and ligaments very much diseased. Bone sections healthy | Entirely healed. Knee ankylosed, half an inch shortening. Walks well | 1877 : Disappeared from observation for two years, during which time had been able to walk and attend school perfectly well. Lately, the leather splint, with which she went out, having become much too short and small, has permitted a slight bowing outwards of the whole leg; in which curvature the tibia and the femur partake. (Rickety.) Shortening has increased, but its extent uncertain.<br>1880 : Knee perfectly straight, can walk so well that the excision is scarcely noticeable. Slight shortening; wearing high-heeled boot | — |
| Patella much eroded; some bony ankylosis between internal condyle of femur and internal tuberosity of tibia. Large sequestrum in external condyle, extending into joint. Cheesy material in both tibia and femur. The whole of both epiphyses were cut away in order to shorten the limb | Hip amputation healed, knee excision healed and can bear weight to a certain extent on it with crutches. General health immensely improved, has become quite a lively wiry lad | This lad wrote to me from a Home for Crippled Boys in 1883, saying that he was quite well, but that the left had become contracted to a certain extent | — |
| Enormous mass of pulpy material on inner side of femur extending up about three inches, dissected off. Suppuration all round joint, chiefly in front and behind. Cartilages disappearing; bone sections healthy | One sinus in stump not quite healed, general health very much improved | Not since heard of | Satisfactory union after excision did not take place, and it is possible that the dissecting off so much pulpy periosteum may have caused the necrosis which subsequently occurred. |
| Complete disorganisation. Extreme flexion. Evident lengthening of the condyles of the femur. 4¾ inches of bone cut away | Bones ankylosed. No sinuses. 4½ inches shortening. Can bear weight on it well | A twelvemonth after leaving hospital wrote to me to say that he could do a day of 12 hours' agricultural work "as well as the best of them." Jan. 1, 1885 : Leg straight, sound, and strong. Six inches shortening, has been working regularly as a labourer since the operation | In spite of the amount of bone cut away, it was found impossible to get the limb perfectly straight, and a slight amount of displacement afterwards occurred, which, however, only *retarded* the cure. |

| No. | Initials, sex, and age. | Disease. | Duration of disease before excision. | Previous treatment. | Admission and discharge. | Duration of stay in hospital. | Operations performed. |
|---|---|---|---|---|---|---|---|
| 28 | J. C., F., 46 | Hydatid disease of lower end of femur | 2 years | Nil. Walked with crutches for 10 months | A. Sept. 1, 1875<br>D. Feb. 29, 1876 | 6 months<br>After excision 5½ months | Excision Sept. 10, 1875 |
| 29 | P. B., F., 8 | Disorganisation of left knee, with necrosis of tibia. Abscess in popliteal space healed | 3 years | Rest | A. Sep. 15, 1875<br>D. July 7, 1876 | 10 months<br>After excision 9¾ months | Excision Sept. 21, 1875. Incision into sinus in popliteal space Mar. 21, 1876 |
| 30 | A. T., M., 28 | Osteo-arthritis of right knee, ditto of right shoulder with complete wasting of deltoid. Some symptoms of phthisis | 19 mos. | Blisters. Iodine. Poultices. Enforced rest | A. Oct. 27, 1875<br>D. June 1, 1876 | 7 months<br>After excision 6 months | Excision Nov. 27, 1875 |
| 31 | J. F., M., 22 | Hydrarthrosis with disorganisation of left knee | 2 years | Rest, with splints, strapped with Ung. Hyd. In-patient at provincial hospital for 3 mos. Out-patient at Guy's for do. | A. Nov. 8, 1875<br>D. June 17, 1876 | 7¼ months<br>After excision 6¾ months | Joint tapped Nov. 17, 1875. 1¼ pints serous pus withdrawn. Excision Nov. 22, 1875. Hydrarthrosis sac incised on Feb. 2, 1876, and again on Feb. 22nd |

| State of joint at time of excision. | State on discharge. | After history. | Remarks. |
|---|---|---|---|
| Much swelling and fluid in joint, which grated on movement. Articular cartilages everywhere gone, and bone surfaces exposed. Hydatid cysts attached to lower end of femur in great abundance. External condyle hollowed out by them | Knee ankylosed. No sinuses. Bears weight well upon it | Came to hospital in Aug., 1877. Walking well, and able to do her household work perfectly. Very slight shortening<br>Jan. 1, 1885: Leg straight, sound, and strong. About 1½ inches shortening | Case gave much trouble after the operation by the deluges of serous discharge which poured away through the gauze dressing, and which continued for over one month. This became fœtid within an hour or two of reaching the external air, but in spite of this the wound never became foul,—the gauze itself and the discharge within the wound remaining always sweet. |
| Much pulpy material. Cartilages absorbed. Femoral bone section healthy. Ditto tibial, but behind it, hollowing it out, a great abscess cavity containing a pyramidal sequestrum 2 inches long | Firm (fibrous ?) ankylosis. 1½ inches shortening. One or two sinuses not yet healed. Rests weight a little upon limb, but cautiously, mostly with crutches | Heard of frequently. Sinuses healed. Limb straight. Walks well upon it.<br>Jan. 1, 1885: "Limb strong and sound, but slightly flexed. About three inches shortening" | Case remarkable for the extent and form of the sequestrum. |
| Ligaments and inter-articular cartilages vascularised and softened. Polypoid growths (not bony) from the synovial membranes. Articular cartilages flaking off ends of bone (epiphysial ostitis). Caseous bone in tibial section | Bones ankylosed. Can bear weight on limb, but one or two superficial sores remained | Seen twice or thrice. Very great improvement in general health. Walks well and does his business | Bedsore formed two days after excision, partly from his helplessness. Nevertheless, wound was healed in 5 weeks. From an accidental strain he partly broke it open again, and owing to the state of his general health, it absolutely refused to heal again while in hospital, though kept in mainly on this account. Healed very shortly after he left hospital. |
| Filled with sero-pus. Softening and vascularisation of the ligaments and synovial membrane. Caseous bone in external condyle and tuberosity | Firmly ankylosed. No sinuses. Walking with crutches | Seen once or twice since. Walking well on leg, which is perfectly straight, firmly ankylosed and serviceable. Shortening three quarters of an inch | Excision wound healed 5 weeks after operation. Prolonged stay in hospital due to the necessity of curing the immense sac on front of the thigh. Accomplished by a very long incision reaching from trochanter to knee. Sac lined with polypoid granulation growths and caseating pyo-genic membrane. It was in close contact with the bone underneath the extensor muscles. All contents cleared away and sac made to granulate from bottom. |

| No. | Initials, sex, and age. | Disease. | Duration of disease before excision. | Previous treatment. | Admission and discharge. | Duration of stay in hospital. | Operations performed. |
|---|---|---|---|---|---|---|---|
| 32 | G. B., M., 20 | Strumous arthritis | 4 years | Rest, with strapping, ointment and iodine | A. Jan. 1, 1876<br>D. July 18, 1876<br>Re-admitted Sept. 4, 1876<br>Re-discharged Nov. 30, 1876 | 6½ months.<br>3 months<br>After excision 6½ months.<br>After amputation 5 weeks | Excision Jan. 8, 1876. Sinuses sponged out with $HNO_3$. Amputation Oct. 20, 1876 |
| 33 | C. B., M., 2½ | Pulpy, left knee, but child very eczematous, and with some evidences of C. S. | 10¼ mos. | Rest for 6¾ months. Incisions | A. May 1, 1875<br>D. May 19, 1876<br>Re-admitted Feb. 21, 1877<br>Re-discharged April 20, 1877 | 12½ mos.<br>2 mos.<br>After excision 6 mos.<br>After amputation 6 weeks | Excision Nov. 22, 1875<br>Amputation March 5, 1877 |
| 34 | S. N., F., 4 | Pulpy, right knee. Much eczema | 2¾ years | Rest and extension for 3 months. Biceps tendon divided | A. June 3, 1875<br>D. Oct. 11, 1876 | 1 year 4 months.<br>After excision 1 year | Excision Oct. 18, 1875 |
| 35 | L. P., F., 9 | Pulpy, right knee | 4 years | Has been in hospital three times previously. Treated with rest, splints, and extension. Plaster bandages applied on each occasion when discharged | A. Apr. 29, 1876<br>D. Sept. 21, 1876 | 5 months<br>After excision 4 months | Excision May 25, 1876 |

| State of joint at time of excision. | State on discharge. | After history. | Remarks. |
|---|---|---|---|
| Backward and outward dislocation. Not much swelling. Vascularisation and softening of all the ligaments, and inter-articular cartilages. Caseous bone in tibia | On July 18th ankylosis of part of the bone sections, but a sinus extending between them at another point. Some bare bone perceptible. Sent into country. On Nov. 30; stump healed | Walking well on artificial leg | It was found impossible to close the sinus between the bones, though sponged out more than once with strong nitric acid. Hence, although firm ankylosis had taken place at one part, it was thought best to amputate the leg. |
| Numerous sinuses from recent abscesses in joint. Much pulp. Great destruction of cartilages, and epiphysial nuclei reduced to jelly | Fibrous union. Two or three sinuses remaining, in connection with masses of pulp. Great improvement in child's health. Eczema and ophthalmia disappeared<br><br>Stump healed. State of health still further improved | Seen 2 months after final discharge. Improvement continued. (From Union) | No father; mother ran away, so that the boy had to be taken to the Union after the excision. As it was impossible to give the requisite amount of care there to such a case, it was thought best to amputate through the thigh. There was found fibrous union between the ends of the bone, and the sinuses were only in connection with masses of pulp. In a well-ordered family this limb would most likely have been saved. |
| Abscess in joint. Both femoral and tibial bone sections caseating | Can walk without crutches. Two or three sinuses left. Sent to country | On Jan. 1, 1877, she was running about well without crutches. Sinuses quite healed. Seen once since. Remains in good condition. Jan. 1, 1885: "Knee bent at angle of 45°; left leg 23", right leg 24½"; bones ankylosed; limb sound" | — |
| Right knee pulpy. Backward and outward dislocation; abscess over outer tuberosity, and another over outer condyle. Joint partially obliterated. Bone sections fatty, but otherwise healthy. Posterior part of femoral surface bare | Walks on crutches. Two or three sinuses existing. In good health | Seen July 7, 1877. Has been walking to school well upon the leg since Christmas; two superficial sores, however, still remain open. Leg straight. March 22, 1879: Wears leather splint; sinuses have healed; foot slightly inverted; no shortening apparent. Nov. 27, 1880: 1" shortening; leg straight; no splint now | — |

| No. | Initials, sex, and age. | Disease. | Duration of disease before excision. | Previous treatment. | Admission and discharge. | Duration of stay in hospital. | Operations performed. |
|---|---|---|---|---|---|---|---|
| 36 | E. H., F., 6 | Pulpy, right knee | 15 mos. | Unknown. From country (Aldershot) | A. Nov. 29, 1875<br>D. June 21, 1876<br>Delayed by attack of whooping-cough | 7 months<br>After excision 6 months<br>After amputation 5½ mos. | Excision Dec. 21, 1875. Amputation Jan. 3, 1876. Ring of necrous bone removed from stump after the whooping-cough on May 8, 1876 |
| 37 | E. J., F., 8 | Extreme genu valgum, with rickety femora | Since first able to walk | Splints | A. Oct. 14, 1876<br>D. May 1, 1877 | 6½ months<br>After excision 6 months | Excision of left knee Oct. 30, 1876: Splints to right knee |
| 38 | E. P., M., 5 | Pulpy, right knee | 2 years | Splints; hamstring tendons divided. Twice in hospital | A. Feb. 16, 1876<br>D. Aug. 4, 1876 | 5¾ months<br>After excision 4¾ months | Excision March 14, 1876 |
| 39 | W. R., M., 18 | Pulpy disease of left knee. Dislocation, grating. 2¼ inches larger round than right | 14 mos. | Rest, splints, Ung. hyd. and iodine | A. Feb. 29, 1876<br>D. Sept. 19, 1876 | 6¼ months<br>After excision 6¼ months | Excision March 7, 1876.<br>Pulpy swelling about joint incised April 2, 1876 |
| 40 | E. S., M., 11 | Pulpy, left knee | 8 years | Splints. Extension. Was in hospital once previously; as an out-patient many times | A. Apr. 12, 1876<br>D. Sept. 17, 1876 | 5 months<br>After excision 4 months | Excision March 22, 1876 |

| State of joint at time of excision. | State on discharge. | After history. | Remarks. |
|---|---|---|---|
| Cartilages roughened and flaking off. In femoral section caseous pus. Ligaments converted into pulp | Stump healed | Unknown | — |
| Internal condyle and tuberosity very much elongated, so that section of the bone was difficult. Vascular synovial membrane encroaching on articular cartilage | Firmly ankylosed. Right knee not so straight as the left | Continues to walk well.<br>Jan. 1, 1885: Leg flexed, but sound and strong. 3 inches shortening | — |
| Bone sections healthy; much pulpy disease of synovial membrane and capsular ligaments. Inter-articular cartilage gone, and articular cartilages much eroded | No sinuses. Fibrous ankylosis | Not since heard of | — |
| Very much pulpy material, caseating in several places. Joint full of pus and broken-down pulp. Some caseating masses in tibial and femoral bone sections. Backward dislocation not great; cartilage eroded, causing grating | Wound ankylosed. One or two sinuses remaining, which had remained in same state for some time, refusing to heal. Patient's health suffering from confinement | Great and speedy improvement in general health after his return home into the country.<br>Heard of in summer of 1877 (by letter): leg completely healed; can walk two miles actively without pain.<br>Jan. 1, 1885: Leg sound, straight, and strong; no shortening. Had spinal caries, with two psoas abscesses discharging for two years; these have now been healed for four years. | — |
| Much swelling; some dislocation; synovial membrane very vascular, and encroaching on cartilages. Semilunar cartilages much diseased. Bone sections healthy | Entirely healed; bones ankylosed. Very slight shortening (¼ in.). No sinuses. Walks well | Seen Sept., 1877. Continues perfectly well, and able to use his leg with hardly a perceptible limp.<br>Jan. 1, 1885: "Limb sound and strong, very slightly flexed; less than ¼ inch shortening" | Patient's stay in hospital very much prolonged by a very severe attack of eczema which he had on the affected leg after the excised wound had entirely healed. Cicatrisation of the wound was complete within five weeks after the operation. |

| No. | Initials, sex, and age. | Disease. | Duration of disease before excision. | Previous treatment. | Admission and discharge. | Duration of stay in hospital. | Operations performed. |
|---|---|---|---|---|---|---|---|
| 41 | W. C., M., 2½ yrs. | Pulpy, left knee, with caseous osteitis | 17 mos. | Knee strapped and painted. Splints. Poultices | A. May 31, 1876<br>D. Oct. 27, 1876 | 5 months<br>After excision 3 months | Excision July 25, 1876 |
| 42 | A. N., F., 6 | Pulpy, right knee | 2½ years | Splints, starch bandages, iodine, and medicine. Has been twice in different hospitals, and has also been out-patient | A. June 7, 1876<br>D. Nov. 6, 1876 | 5 months<br>After excision 4 months | Excision July 7, 1876 |
| 43 | A. B., M., 9 | Pulpy, left knee | 3 years | Rest, with splints. Twice an out-patient for some months | A. Apr. 18, 1877<br>D. About Oct. | — | Excision May 15, 1877<br>Amputation July 27, 1877 |
| 44 | J. C., M., 9 | Pulpy, right knee | 1 year | Back splint. Leather splint. O.P. at Guy's | A. June 16, 1877<br>D. After Sept. | — | Excision July 25, 1877 |
| 45 | J. M., M., 5½ | Pulpy, left knee | 9 mos. | Rest. Extension. In hospital for 2 months previously | A. April 3, 1876<br>D. Jan. 21, 1877 | 9½ months<br>After excision 8½ months | Excision May 9, 1876<br>Nitric acid applied for cure of sinuses on Oct. 13 |

| State of joint at time of excision. | State on discharge. | After history. | Remarks. |
|---|---|---|---|
| Open sinus extending into the joint, discharging much pus. Complete disorganisation. Very large cavity found extending down to the middle of shaft of tibia, filled with caseous pus and granulation | Wound entirely healed; fibrous ankylosis | Feb. 25, 1885: Leg straight, strong, and sound; $2\frac{1}{4}$″ shortening | — |
| Much swelling; $1\frac{1}{2}$″ difference. Articular cartilage mostly gone. Cavities containing pulpy granulation found in the outer condyle of the femur and external tuberosity of the tibia | Entirely healed; can walk on leg. Very slight shortening | Last seen in summer, 1877. Plays and runs about with other children. Frequently heard of since | — |
| Backward and outward dislocation. Good deal of pulpy tissue in synovial membrane and ligaments. Vascular synovial membrane encroaching much on the cartilages | Stump nearly healed Sept. 14, 1877. Very great improvement in general health | — | By accident this patient was not operated on with full attention to antiseptic details; whether from this cause, or from the boy's natural debility, he did not do well. Suppuration occurred. Temperature rose, and it was to save his life that the leg was amputated. |
| Much swelling about joint (1 inch difference), which was flexed at angle of 45°. Backward and outward dislocation. Synovial membrane pulpy, and encroaching upon articular cartilages, which were flaking from subjacent bone. An exposed and carious surface on the tibia. Large abscess cavity in position of semi-membranosus bursa | Sept. 14, 1877: Fibrous union. Some sinuses remaining open. Promises well | — | A very delicate strumous boy, with a phthisical history. |
| Backward dislocation, with some permanent flexion. Moderate amount of pulpy material. Bone sections healthy. Complete disorganisation of ligaments and inter-articular cartilages. Case ating granulation, and pus extended for 3 inches under quadriceps muscle | Wound healed. Limb firmly ankylosed, but a good deal bowed, and tibia and fibula rotated outwards | — | — |

| No. | Initials, sex, and age. | Disease. | Duration of disease before excision. | Previous treatment. | Admission and discharge. | Duration of stay in hospital. | Operations performed. |
|---|---|---|---|---|---|---|---|
| 46 | R. M., M., 27 | Pulpy, right knee | 6 years | In provincial hospital for 3 months. Blisters, iodine, strapping | A. July 6, 1876<br>D. Mar. 30, 1877 | 8¾ months<br>After excision 8¼ months | Excision July 18, 1876 |
| 47 | J. K., M., 36 | Arthritis of right knee. Chronic osteo-arthritis | 4½ years | Rest with splints and strapping. Iodine. In hospital for 2 months previously | A. Sept. 20, 1876<br>D. April 7, 1877 | 6½ months<br>After excision 6¼ months | Excision Sept. 20, 1876 |
| 48 | C. M., M., 43 | Gunshot wound of right knee-joint | 4 hours | None | A. April 16, 1877<br>Died April 21, 1877 | 5 days<br>After excision 5 days | Excision April 16, 1877 |

| State of joint at time of excision. | State on discharge. | After history. | Remarks. |
|---|---|---|---|
| Much pulpy swelling; tibia and fibula displaced backwards. Pulpy cavity in external tuberosity. Caseous bone in inner tuberosity. Similar conditions in condyles of femur | Fibrous union. Small discharging sinus; but walks well with the leather splint on | Has continued to improve. Leg becoming much stronger since leaving the hospital. Ankylosis never complete | Ossific union did not take place, owing apparently to the skin becoming inverted between the bones. This was destroyed once or twice with strong nitric acid, and the gap very much diminished, but still ossification did not take place. Further treatment was proposed; but the patient being able to walk well on leg with a leather splint on, declined to have anything further done.<br>Heard on Nov. 20, 1877, from Mr. Fry, who sent him up, that he was earning 25s. a week as engine driver; that he still wore the leather splint, but that with it the limb seemed quite strong and useful.<br>Feb. 15, 1884: Heard from Mr. Latimer that he had amputated through the thigh owing to suppuration along line of old excision. He had worn a leather splint for over seven years, and this had worn out and not been replaced. The increased strain on the knee had been the cause of fresh suppuration. Bones normal. |
| Ligaments and interarticular cartilages vascularised and softened. Articular cartilages considerably eroded. Bone sections healthy | Firm ankylosis. One or two superficial sores not quite healed. Can walk well | Seen in July. Leg quite healed. Can use it well.<br>May 26, 1878: Walks twenty miles without fatigue; limb straight and strong; able to work well. Wearing leather splint.<br>Jan. 1, 1885: "Leg straight, strong, and sound, about $\frac{3}{8}$" shortening" | Patient's convalescence retarded by a slight slipping in the position of the bones, which took place in the first 24 hours, which gave rise to some suppuration, and by an attack of erysipelas which took place after the antiseptic dressings were left off. |
| All the front of joint blown away. Comminuted fracture of patella. Shot lodged in condyles of femur. After excision a gap of 3 inches was left in the skin in front of the joint on account of the great loss of soft parts | Death from jaundice, patient having been a very intemperate man. P.M.—Fatty liver. Bones healthy. Periosteum and vessels for 2" infiltrated with pus | Though this was a death *after* excision, it would be plainly unfair to consider it a death *from* excision | Patient was slightly jaundiced on admission. Consent to immediate amputation was refused by the friends, who would only permit excision. He did not rally after taking $CHCl_3$ to have the splint changed. |

| No. | Initials, sex, and age. | Disease. | Duration of disease before excision. | Previous treatment. | Admission and discharge. | Duration of stay in hospital. | Operations performed. |
|---|---|---|---|---|---|---|---|
| 49 | J. B., M., 51 | Osteo-arthritis of right knee | 8 mos. | Rest, cold-water bandages, splints, &c. Once in hospital previously | A. Jan. 31, 1877<br>D. May 17, 1877 | 3½ months<br>After excision 3¼ months | Excision Feb. 6, 1877 |
| 50 | D. G., M., 6½ | Pulpy, right knee | 5½ mos. | Rest by extension and splints | A. Jan. 22, 1877<br>D. June 29, 1877 | 5¼ months<br>After excision 2½ months | Excision April 10, 1877 |
| 51 | S. S., F., 11 | Double genu valgum. Double talipes varus. Infantile paralysis of lower limbs, so that she cannot use the extensors, which do not respond at all to the continuous current | Since baby-hood | Tendo Achillis divided on both sides. Splints. Galvanism | A. June 8, 1876<br>D. June 30, 1877 | 12¾ months<br>After 1st excision 9½ months<br>After 2nd excision 6¼ months | Excision of both knees; right Sept. 19, 1876; left Jan. 5, 1877 |
| 52 | A. C., F., 6¾ | Pulpy right knee | 7 mos. | Rest | A. April 11, 1877<br>D. Aug. 2, 1877 | 3¾ months<br>After excision 2¾ months | Excision May 14, 1877 |
| 53 | E. D., F., 45 | Pulpy, left knee. Has been confined in lunatic asylum some years ago for an attack of melancholia | 21 mos. | Rest. Counter-irritants | A. July 17, 1877<br>D. Dec. 15, 1877 | 5 months | Excision July 23, 1877 |

| State of joint at time of excision. | State on discharge. | After history. | Remarks. |
|---|---|---|---|
| Swelling; ¾″ difference. Bone sections healthy. Semilunar cartilages vascularised and softened. Large mass of caseous material underneath quadriceps. Articular cartilages encroached upon and eroded by the swollen synovial membrane | Wound healed. Bones ankylosed. Can bear weight upon it, though he is somewhat timid | Continued to improve | — |
| Much swelling. 1 inch difference. Much pulpy material. Bone sections healthy, but articular cartilages eroded and ligaments softened | Walks about the ward, knee healed entirely | June 30, 1881: Fell off cart and fractured left leg near line of union. Great effusion of blood into tissues above and below<br>Aug. 11, 1881: Went home with bones firmly united. | — |
| Knees healthy, but articular cartilage rather encroached upon at the margin from disuse | Both knees ankylosed so that she can bear weight upon them. Gets up- and down-stairs. The talipes much corrected, but position of foot still by no means perfect | Jan. 1, 1885: Right leg much flexed, but sound and strong, but does not feel so. 2 inches shortening, wearing splint. Left leg straight and strong. The patient usually walks half a mile in the day, using 2 sticks. The left foot is inverted | Both excision wounds healed primarily |
| Swelling 1¼ inches. Pretty free motion. Joint completely disorganised. All the soft parts infiltrated with pulpy material. Bone sections healthy | Wound entirely healed. Can bear weight on limb | — | – |
| Much swelling, 2½ inches difference. Ligaments vascularised and soft. Semilunar cartilages partly absorbed. Much caseous material beneath quadriceps. Bone sections healthy | Hysterical. Complains of pain in knee. Able to walk with crutches. Wearing leather splint. There is a little movement between bones | — | Healed in five weeks after operation. |

| No. | Initials, sex, and age. | Disease. | Duration of disease before excision. | Previous treatment. | Admission and discharge. | Duration of stay in hospital. | Operations performed. |
|---|---|---|---|---|---|---|---|
| 54 | H. K., F., 17 | Pulpy, right knee | 2 years. Worse last 2 mos. | Rest. Iodine applications | A. June 11, 1877<br>D. May 23, 1878<br>Re-admitted Nov. 6, 1878<br>D. Feb. 18, 1879 | 11 months<br>3 months | Excision June 19, 1877. Oct. 7, 1877: Nitric acid applied to sinuses. Jan. 8, 1878: Abscess opened on inner side of knee. Mar. 26, 1878: Amputation through lower ⅓ thigh, antero-posterior flaps. April 4, 1878: Abscess in Scarpa's triangle opened |
| 55 | J. B., M., 6½ | Pulpy, right knee | 10 mos. | Splints. Bandaging. Poulticing. No medical advice lately | A. May 12, 1877<br>D. Nov. 18, 1877 | 6 months | Excision June 1, 1877 |
| 56 | M. S., F., 6 | Pulpy, left knee | 21 mos. | Attended at a dispensary 6 months. Iodine applied. Medicinal internal treatment. Out-patient at Guy's Hospital a month. Ung. Hyd. Co. and splint applied. Not better. Admitted July 10, 1876. Rest. Extension. Medicinal. Discharged August 4, 1876, wearing a Bavarian splint | A. July 23, 1877<br>D. Nov. 22, 1877 | 4 months | Excision July 26, 1877 |

| State of joint at time of excision. | State on discharge. | After history. | Remarks. |
|---|---|---|---|
| Great swelling. Difference 4¾ inches. Skin tense and rigid. Doughy fluctuation on inner side. Joint filled with caseating pus and granulation, which extended for 4 or 5 inches upwards underneath the quadriceps. Ligaments and interarticular cartilages destroyed. Bone sections healthy but fatty | — | Nov. 6, 1878: Readmitted. The stump has not healed. Sequestra were removed and sinuses opened. Went home Feb. 18, 1879, with small granulating wound.<br>June 23, 1881: Patient is now quite well, and has a good situation | Very cachectic girl with ozæna; altogether an exceedingly unfavorable case from the great extent of the mischief, but one which healed up in about ten weeks without a single bad symptom. On Nov. 22, 1877, patient was able to get about the ward. Suppuration soon, however, recurred round the ends of the bones, and amputation became necessary in March, 1878. |
| Much swelling, 2 inches difference. Joint containing sero-pus. Ligaments vascularised and softened. Articular cartilages nearly disappeared. Bone sections healthy | Wound healed six weeks after operation, but some projection appearing from the external condyle, dresser bandaged it very tightly to try and rectify it, and this caused the skin over it to slough. Leg straight, able to walk without crutches | Dec. 30, 1877: Still small sinus.<br>Oct. 28, 1878: ⅞″ shortening? slight antero-post. bowing. Wound healed, can walk well—wears leather splint.<br>March 4, 1881: Limb very serviceable, not quite straight, bones united at angle of 170°. Plaster-of-Paris splint applied instead of leather one.<br>Jan. 1, 1885: Leg bent at angle of 120°. No shortening of bones, limb strong and sound | — |
| Dislocation backwards. Large caseating mass beneath quadriceps. Caseating patch in tibial section and similar smaller one in femur. Synovial membrane and ligaments quite disorganised | Excision wound entirely healed | Seen on Dec. 12, 1877. Doing exceedingly well. Slight superficial sore formed over cicatrix from rubbing of leather splint | — |

| No. | Initials, sex, and age. | Disease. | Duration of disease before excision. | Previous treatment. | Admission and discharge. | Duration of stay in hospital. | Operations performed. |
|---|---|---|---|---|---|---|---|
| 57 | J. K., M., 7 | Caseous disease of right os calcis. Pulpy, left knee | 15 mos. | In Lydia, one year ago. Splint. Oleate of mercury. Leather splint. Not cured after | A. Feb. 19, 1877<br>D. Oct. 24, 1877 | After excision 6 months | For necrosed bone in right os calcis on March 6, 1877. Excision of left knee on April 27, 1877 |
| | 2 months' stay. In August, 1876, right ankle swelled; in Oct., 1876, admitted to St. Bartholomew's Hospital, stayed 11 weeks. Ankle operated on. Wound never healed. Cod-liver oil and steel | | | | | | |
| 58 | S. C., M., 9 | Atrophy of limb. Pulpy, right knee. Stretching of ligaments and almost complete inability to support himself on the side. 1½ inches shortening. Infantile paralysis | From infancy 6 mos. old | Has had almost everything tried as out-patient and in-patient at a metropolitan hospital | A. June 7, 1877<br>D. Sept. 14, 1877 | After excision 3 months | Excision June 15, 1877 |
| 59 | W. V., M., 13 | Acute pulpy, left knee | 7 wks. | Rest, with splints | A. May 9, 1877<br>D. Oct. 24, 1877 | 5 months | Excision May 22, 1877 |
| 60 | C. W., M., 3 | Pulpy, left knee | 10 mos. | Ointment and back splint | A. Feb. 7, 1877<br>D. June 3, 1877 | 4 months | Excision March 13, 1877 |

| State of joint at time of excision. | State on discharge. | After history. | Remarks. |
|---|---|---|---|
| Not much displacement or swelling. Joint completely disorganised. Caseous bone and granulation extended past the epiphysial line 2 inches into the shaft; cleared out | Still in, Sept. 18, 1877. Knee wound nearly healed. Sinus remains over os calcis and some more bare bone has been detected in it. Great improvement in boy's general health | Feb. 13, 1878: Legs of about equal length, straight; able to walk without crutches. Leather splint | — |
| Quiescent. Full flexion and extension difficult, but ligaments so loose as to permit much lateral movement. Joint cavity obliterated, replaced by connective tissue, but articular cartilages present, except at posterior part of tibia, where the bone was bare. Condyles of femur showing two centres of ossification, separated by a deep band of articular cartilage. | Quite healed. Firm (fibrous ?) ankylosis. Walks well with boot, $1\frac{3}{4}$ inches deep in sole | — | — |
| Considerable swelling. Fluctuation. Joint full of sero-pus. Much pulpy material about the synovial membrane, which had encroached considerably upon the articular cartilage | Excision wound healed. Very fair ankylosis. | Nov. 27, 1877: Good straight limb. No tenderness | Much swelling occurred in the soft parts immediately after the operation, and somewhat retarded the rapidity of the cure, but this did not affect his general health in the slightest degree, the usual temperature being 99°, and having never once risen above 100°. |
| Caseous bone in inner condyle with superficial abscess. Pulpy synovial membrane encroaching on all cartilages. Joint red and swollen | Limb straight, some movement between bones. Leather splint | Nov. 6, 1878: Union firm, slight bending. $\frac{3}{4}''$ shortening.<br>June 5, 1882: Leg bent slightly outwards and flexed with inversion of foot; can run about and does not tire readily.<br>Feb., 1885: Leg bent at angle of 120°. $2\frac{1}{2}''$ shortening. Quite strong and sound | Child had ophthalmia and otorrhœa afterwards, but wound healed in 5 weeks. |

| No. | Initials, sex, and age. | Disease. | Duration of disease before excision. | Previous treatment. | Admission and discharge. | Duration of stay in hospital. | Operations performed. |
|---|---|---|---|---|---|---|---|
| 61 | M. R., F., 7 | Pulpy disease, right knee | 12 mos. | Extension with weights. Rest in bed 3 months | A. April 16, 1878<br>D. Dec. 18, 1878 | 8 months<br>5 months after excision | Excision July 22, 1878. Nitric acid to sinuses Dec. 6. Sept. 21 : Pulpy material scraped from intra-condyloid pouch |
| 62 | J. M., M., 32 | Pulpy disease, left knee | 4 years | Blistered, &c. | A. May 8, 1878<br>D. Nov. 27, 1878 | 6 months | Excision May 21. 2 sinuses slit up July 22. Caseating material removed from inner side of knee, Sept. 21. Amputation through thigh, Oct. 15 |
| 63 | J. C., M., 18 | Pulpy disease, right knee | Gradually worse since rheumatic fever 12 yrs. ago | Back splint | A. July 3, 1878<br>D. Oct. 17, 1878 | 3 months | July 19 : Excision |
| 64 | F. R., M., 3½ | Pulpy, right knee | 2 mos. | Extension and rest | A. Apr. 1, 1878<br>D. Sept. 20, 1878 | 5 months | May 17 : Excision. Sept. 5 : Abscess on outer side opened. April 4, 1879 : Amputation through lower ⅓ femur |
| 65 | H. L., F., 30 | Partial ankylosis, left knee | 8 yrs. unable to flex knee. Inflamed 7 wks. | — | A. June 5, 1878<br>D. Sept. 6, 1878 | 3 months | Excision June 14, 1878 |

| State of joint at time of excision. | State on discharge. | After history. | Remarks. |
|---|---|---|---|
| Knee immoveably flexed at right angles. Sinuses on outer side since February. Tibia dislocated back. Outer tuberosity of tibia. Diseased condyles of femur eroded | — | Jan. 1, 1885: Flexed | Old left hip disease. |
| Imperfect and painful movement. Head of tibia diseased | — | — | — |
| Tibia dislocated backwards. Knee hot and swollen. Movement limited and painful. Pus in joint. Patella dislocated outwards. Removed caseous bone in ends of femur and tibia | Wound healed. Leather splint. Good position | Nov. 18, 1878: Leg in good position | — |
| Joint painful. Motion limited. Caseating pulpy material in joint | Sinuses still present | April 4, 1879: Readmitted. Child is in bad health, and has numerous sinuses round excision cicatrix. Amputation through thigh at lower third. Bones in good position. Epiphysial growth-lamellæ untouched, but bone between them was caseous and soft. Caseous pulpy granulations at back of joint.<br>April 14, 1879: Caries of left tarsus; abscess opened.<br>May 23, 1879: Pirogoff's amputation of left foot.<br>July 2, 1879: Went home. Both stumps healed | Knee incision healed by first intention, but some pus was retained on outer side. |
| Knee painful and inflamed. Old cicatrix on outer side. Patella adherent to femur | Wound healed | Jan. 1, 1885: Limb sound and strong, slightly flexed and shortened | — |

| No. | Initials, sex, and age. | Disease. | Duration of disease before excision. | Previous treatment. | Admission and discharge. | Duration of stay in hospital. | Operations performed. |
|---|---|---|---|---|---|---|---|
| 66 | H. L., F., 19 | — | Knee always has been troublesome since 3 mos. old. Worse 3 mos. | — | A. Jan. 16, 1878<br>D. July 4, 1878<br>Re-admitted July 29, 1878<br>Death Aug. 14, 1878 | — | Excision Jan. 29, 1878<br>Amputation July 29, 1878 |
| 67 | S. D., F., 9 | Pulpy, left knee | 2 years | Extension with weights for many months | A. May 8, 1878<br>D. Aug. 3, 1878 | 3 months | Excision May 28 |
| 68 | W. W., M., 36 | Pulpy, right knee | — | — | A. Oct. 24, 1877<br>D. Mar. 12, 1878 | 5 months | Exploratory puncture Nov. 2<br>Excision Nov. 16.<br>Dec. 17: Abscess opened |
| 69 | E. B., F., 14 | Extreme genu valgum of left leg. Infantile paralysis (?). Pulpy disease of knee. Great wasting of muscles of limb. Foot everted | Since infancy | Plaster-of-Paris splints | A. Nov. 11, 1878<br>D. Feb. 19, 1879 | 3 months | Excision Nov. 19, 1878 |
| 70 | M. R., F., 12 | Pulpy, left knee | 15 mos. | Rest, lotions | A. April 16, 1879<br>D. Sept. 23, 1879 | 5 months<br>4½ months after excision | Excision May 6, 1879 |

| State of joint at time of excision. | State on discharge. | After history. | Remarks. |
|---|---|---|---|
| Knee painful, hot, partially flexed; caseating bone with chronic osteitis of head of tibia. All cartilages somewhat eroded. Exostosis from outer condyle of femur | Unsatisfactory. Amputation advised, but parents refused to allow any further operation | July 29: Re-admitted. Thigh amputated at lower $\frac{1}{3}$. Developed tubercular meningitis, and died Aug. 14 | June 10: Cough, night sweats, &c. Phthisis, both apices. |
| Knee rigidly flexed at a right angle. Tibia dislocated backwards. Bones healthy. Cartilages eroded. Pulpy synovial membrane | $\frac{3}{4}$ inch shortening. Wound healed. Leg straight | — | — |
| Knee swollen, especially over head of tibia. Pus in joint. Extensive disease of joint | — | Aug. 22, 1878: Leg strong and straight. Able to walk 3 miles to his work, and has no pain with knee. Jan. 1, 1885: Limb straight, sound, and strong, 1″ shortening | — |
| Patella displaced outwards. Inner condyle of femur much hypertrophied. Membrane diseased. Cartilage eroded. External condyle a mere scale of bone. Crucial ligaments gone | Wound healed | July 26, 1881: Limb firm and straight. 3″ shortening | — |
| Synovial membrane pulpy | Bones united, but there are granulating surfaces on front and outer part of knee | Readmitted Jan. 2, 1881: Two abscesses formed and broke, one on outer side of old incision, and the other 2″ below groin: there is 1″ shortening | Feb. 9, 1881: Patient went home with both surfaces nearly healed. |

| No. | Initials, sex, and age. | Disease. | Duration of disease before excision. | Previous treatment. | Admission and discharge. | Duration of stay in hospital. | Operations performed. |
|---|---|---|---|---|---|---|---|
| 71 | W.W., M., 19 | Pulpy, left knee. Spinal caries, dorsal region | Injury 4 yrs. ago. In bed 18 mos. | Rest in bed | A. Mar. 14, 1878<br>D. Aug. 6, 1879 | 17 months | Excision 1st week in April, 1879 |
| 72 | W. M., M., 15 | Pulpy, right knee lower end of femur. Able to get about till 2 months ago | 6 yrs. ago osteitis of | Rest and splints | A. April 24, 1879<br>D. Aug. 21, 1879 | 4 months | Excision May 20, 1879 |
| 73 | A. W., M., 5 | Pulpy, left knee. Right hip-joint disease | Injury 1 year since | Rest | A. Oct. 13, 1878<br>D. Aug. 1, 1879 | 10 months | Excision Dec. 13, 1878<br>Abscess opened on inner side of knee Feb. 10, 1879<br>Amputation left thigh, lower ⅓, March 22, 1879 |
| 74 | C. T., M., 7 | Pulpy, right knee | Injury 2 years ago | Splints for a few weeks at first. On admission extension and rest | A. Aug. 27, 1879<br>D. Dec. 18, 1879 | 4 months | Excision Sept. 16, 1879 |
| 75 | E. T., F., 20 | Pulpy, right knee | 2 years | Rest and splints for 7 months | A. Mar. 12, 1879<br>D. July 7, 1879 | 4 months | Excision March 25, 1879 |

| State of joint at time of excision. | State on discharge. | After history. | Remarks. |
|---|---|---|---|
| — | Wound healed. Union between bones not quite consolidated | Died Mar. 26, 1880, probably from lardaceous disease | On admission: Right knee flexed immoveably at right angles, partially dislocated backward and outwards. Bedsores on nates. Urine 1015, trace of albumen. Several Sayre's jackets were applied. His long stay was chiefly for the sake of his spinal caries. |
| Erosion of cartilage of femur | Leg straight, but union not perfect, on July 31, 1879 | Jan. 1, 1885: Leg straight, sound, and strong. No shortening | — |
| Knee swollen. Synovial membrane pulpy. Cartilage of patella and femur eroded. Patella in two pieces. Caseous melon-seed bodies in joint | When he went out the stump was sound, and the right hip gave no pain | Re-admitted June 15, 1880, for right hip disease | Amputation was performed as the sinuses were not healing, and led down to ends of bones, which were united by fibrous tissue. Bones healthy. |
| Knee flexed, almost immoveable. Patella adherent to outer condyle. Synovial membrane pulpy. Cartilages eroded. Crucial ligaments gone | Wound healed | April 6, 1881: Has worn leather splint. Limb has been quite sound till 1 month ago, when an abscess began to form in popliteal space. Re-admitted. ½″ lengthening of right lower limb. Pulpy granulating material and pus evacuated from abscess.<br>June 1, 1881: Went out with leg well.<br>June 7, 1882: Leg strong and straight, and never painful. Applied for treatment on account of trouble in shoulder-joint | — |
| Knee flexed, movement limited. Patella dislocated outwards. Synovial membrane pulpy. Fibrous adhesion. Cartilages fibrous | Wound healed. Patient walks on leg with splint on | Dec. 15, 1879: Shortening ½″; wasting of extensors. Circumference: Right thigh 19″, left 22½″. Leg firm and straight. She is able to be on her legs all day | — |

| No. | Initials, sex, and age. | Disease. | Duration of disease before excision. | Previous treatment. | Admission and discharge. | Duration of stay in hospital. | Operations performed. |
|---|---|---|---|---|---|---|---|
| 76 | T. B., M. 12 | Pulpy and suppurating right knee | 2 years | Poultices to abscesses. Extension with weights | A. June 5, 1878<br>D. Apr. 10, 1879 | 11 months | Excision June 28, 1878<br>Sinuses slit up Dec. 6, 1878<br>Feb. 2, 1879: Sinuses slit up, sequestrum removed |
| 77 | T. H., M., 15 | Suppurating and pulpy, right knee | 2 years | Blisters, splints, and rest 9 months, then much better till lately | A. Jan. 15, 1879<br>D. Apr. 23, 1879 | 3 months | Excision Feb. 11, 1879 |
| 78 | J. H., M., 5 | Pulpy, left knee | Injury 6 mos. ago | Rest | A. Mar. 12, 1879<br>D. Nov. 31, 1879 | 8 months | Excision April 22, 1879<br>July 31, 1879: Chronic abscess over scapula opened<br>Sept. 28, 1879: Amputation lower ⅓ femur, ant.-post. flaps |
| 79 | S. D., F., 15 | Pulpy, left knee | 3 years, worse last month | Rest. Blistering. Back-splint | A. Mar. 22, 1880<br>D. Dec. 5, 1880 | 8½ months | April 16, 1880: Excision<br>July 15, 1880: Abscesses opened in leg<br>Aug. 11, 1880: Abscess opened near knee<br>Oct. 8, 1880: Amputation through lower ⅓, left thigh, ant.-post. flaps |
| 80 | H. B., M., 17 | Pulpy, right knee has at various times been inflamed; has had to walk with two sticks for 7 months | For 2½ years knee | Splint. Iodine. Scott's ointment. Cauterisation | A. July 1, 1880<br>D. Oct. 22, 1880 | 3 months after excision | July 23, 1880: Excision |

| State of joint at time of excision. | State on discharge, | After history. | Remarks. |
|---|---|---|---|
| Pus in joint; cartilages eroded. Synovial membrane pulpy. Large sinus extended 2½″ into femur, lined with caseous bone | Limb in good position. Sinus on outer side of leg not quite healed | Jan. 1, 1885: "Leg strong and sound, slightly flexed; 1″ shortening" | — |
| Pus in joint, chiefly in posterior part. Cartilages eroded. Bones healthy. Synovial membrane pulpy | Union firm and bones in good position | Seen 2 or 3 times since. Limb sound. Jan. 1, 1885: "Leg sound, straight, and strong; no shortening" | Wound healed, and patient up in 7 weeks. |
| Pulpy synovial membrane. Caseous cavity in head of tibia. Joint inflamed and swollen. Tibia slightly dislocated backwards | Stump healed | — | The sinuses about the knee could not be got to heal, which necessitated amputation. The bones were healthy, but the sinuses led down to a quantity of pulpy material. |
| Tibia rotated out, dislocated back, and semi-flexed. Cartilages eroded | Dec. 5, 1880: Stump tender, granulations along edges of flaps | July 15, 1880: Bones firmly united. Oct. 8, 1880: There was caseous material round head of fibula and on surface of tibia on outer side. The union between tibia and femur was bony and fairly extensive, being 1″ across out of 1⅝″ | Incision over patella, which was removed and ends of bones excised. Melon-seed bodies in joint, some pedunculated, some free. |
| Knee flexed at angle of 130°; slight movement; leg dislocated back and rotated out | Oct. 22, 1880: Limb straight and firm. ½″ longer than left leg. Wears leather splint | March 25, 1881: Died at home. The limb was quite sound, with but slight flexion. Cause of death unknown | The wound was entirely healed and bones ankylosed in 7 weeks after the excision. |

| No. | Initials, sex, and age. | Disease. | Duration of disease before excision. | Previous treatment. | Admission and discharge. | Duration of stay in hospital. | Operations performed. |
|---|---|---|---|---|---|---|---|
| 81 | E. S., F., 7 | Pulpy, left knee | Injur. l. knee 18 mos. ago | Knee strapped; has worn irons 8 months | A. Apr. 28, 1880<br>D. Aug. 29, 1880 | 5 months | Excision May 7, 1880 |
| 82 | K. L., F., 15 | Periostitis of right femur | Typhoid fever 6 mos. ago, followed by periostitis of femur | Large abscess opened on inner side of condyle. Extension with weights | A. Aug. 27, 1879<br>D. Jan. 8, 1880 | 4½ months | Excision Oct. 17, 1879 |
| 83 | T. S., M., 9 | Pulpy, right knee | 1 year, in bed some weeks | Rest | A. Jan. 7, 1880<br>D. May 5, 1880 | 4 months | Excision Jan. 20, 1880 |
| 84 | J. W., M., 4 | Acute pulpy knee | 5 mos. | Back-splint | A. Oct. 8, 1879<br>Death May 3, 1880 | — | Excision Feb. 2, 1880 |
| 85 | L. H., F., 22 | Partial ankylosis, left knee | 8 years ago abscess in popliteal space, since which, leg has never been straightened | Abscess opened in popliteal space; rest | A. Dec. 10, 1879<br>D. Mar. 19, 1880 | 3 months | Excision Dec. 19, 1879 |
| 86 | F. S., M., 29 | Old disease with partial ankylosis of right knee | Injured right knee 8 years ago, and again 18 months ago, since which he has required a stick. Injured again 11 days ago | Back-splint; rest | A. May 7, 1880<br>Death May 14, 1880 | 1 week | Excision May 11, 1880 |

| State of joint at time of excision. | State on discharge. | After history. | Remarks. |
|---|---|---|---|
| Knee enlarged, flexed; very slight movement, which, however, causes pain | — | — | July 3, 1880: Wound healed. |
| Circumference of right thigh 3″ above condyle 12″, and on left side 9¼″; leg flexed; movement slight. Synovial fringes enlarged and vascular; eroding cartilages. Shaft of femur in lower third was bent backwards | Wound healed | Jan. 1, 1885: Limb strong and straight; 1 inch shortening | — |
| Right knee slightly flexed; leg wasted | Leg straight and bones firmly united, wound healed | — | Congenital talipes equino-varus, for which he had been operated on at Evelina Hospital. |
| Knee enlarged, movement very limited. Pus in joint-sinuses led to two cavities in head of tibia. Cartilages eroded, membrane pulpy. Caseous patches of osteitis extending 1″ into shaft of tibia were gouged out | P.M. — Bones not firmly united, cavity containing pus in head of tibia | — | April 9, 1880: Symptoms of tubercular meningitis appeared. Child died from this May 3, 1880. |
| Patella ankylosed to external condyle of femur at angle of 135°; tibia dislocated backwards and rotated outwards. Can flex knee slightly. Anterior end of condyles nodular | Patient went home wearing a Bavarian limb, firm and straight, but shortened | Jan. 1, 1885: "Limb straight, sound, and strong. 4 inches shortening" | Patella and an inch of femur removed by operation. Temperature raised 2° or 3° for 3 weeks. |
| Leg flexed at right angle to femur. Slight ankylosis of tibia and femur on inner side. Caseous osteitis in both bones | — | — | $CHCl_3$, and then ether were given at the operation. Uncontrollable vomiting, afterwards ending in death on May 14, 1880. P.M.—Organs healthy. Stomach dilated, death due apparently to exhaustion. |

| No. | Initials, sex, and age. | Disease. | Duration of disease before excision. | Previous treatment. | Admission and discharge. | Duration of stay in hospital. | Operations performed. |
|---|---|---|---|---|---|---|---|
| 87 | J. M., M., 58 | Suppurating osteo-arthritis, left knee | 6 mos. ago injured left knee, worse 2 wks. | Iodine. Rest | A. Feb. 27, 1880<br>D. Nov. 5, 1880 | 8 months | Excision April 6, 1880. Sept. 21, 1880: Abscess in popliteal space opened. Oct. 27, 1880: Abscess on inner side of knee opened. Nov. 2, 1880: Antero-posterior flap amputation through lower one-third left femur. Nov. 5, 1880: Death |
| 88 | J. R., F., 25 | Ununited fracture of left femur into joint | Fractured femur 7 mos. ago | Outside splint | A. Nov. 6, 1878<br>D. Feb. 15, 1879 | 3 months | Nov. 26, 1878: Excision and removal of lower fragment of femur. Liq. Sodii injected between bones on five occasions in June, July, and August; Tr. Cantharides 3ij twice in September. Oct. 14, 1879: Lower end of femur removed and tibia refreshed anteriorly, and attached to flaps of periosteum from femur. Jan. 3, 1880: Thigh amputated in lower one third |
| 89 | N. L., M., 29 | Pulpy, right knee has been swollen and painful 4½ years | Struck right knee with hammer 5 years ago. It | Counter-irritation. Elastic bandage lotions | A. May 31, 1880<br>D. Mar. 11, 1881 | 10 months | June 8, 1880: Excision, caseous bone remaining was gouged out. Aug. 12, 1880: Two sinuses slit up. Sept. 10, 1880: Sinus slit up and scraped. Oct. 12, 1880: Sinus on outer side of thigh scraped out. Jan. 5, 1881: Sinuses on inner side of popliteal space slit up. Jan. 12, 1881: Amputation, lower ⅓rd of femur |
| 90 | C. B., M., 11½ | Pulpy, left knee | Left knee swollen for 3½ years | Rest. Bandaging | A. Mar. 14, 1881<br>D. Sept. 23, 1881 | 6 months | Excision Mar. 29, 1881. Aug. 8, 1881: Sinus slit up and pulpy material removed |
| 91 | F. S., M., 6 | Pulpy, left knee | Injured knee 2 years ago | Rest. Bandages, &c. | A. Aug. 24, 1881<br>D. Nov. 26, 1881 | 3 months | Excision Aug. 30, 1881 |

| State of joint at time of excision. | State on discharge. | After history. | Remarks. |
|---|---|---|---|
| Limb extended, movement very slight. Knee enlarged and inflamed. Caseous pus in joint. Synovial membrane and cartilages diseased | — | — | Suffered from stricture of urethra, which was dilated from No. 2 to No. 8. Free drinker, bad arteries; albuminuria before stricture was dilated. Patient during night after amputation lost almost a quart of blood from oozing from the bone. He died chiefly from exhaustion. At p.m., Bright's disease, adherent pleura and pericardium. |
| Transverse fracture was just above condyles, no union; the fracture between the condyles into joint was united. Sept. 14, 1879: False joint existed between bones | Stump well | — | Too large a gap left between the bones (by the removal of the fractured piece of femur) for successful bony union to take place. The wound healed, but only fibrous union between the bones took place. |
| Joint swollen, cartilage bare over inner condyles. Caseous patches in inner condyles of both bones; raised hard margin round patella as in osteo-arthritis. Synovial membrane pulpy and caseous in spots. Melon-seed bodies in joint | March 11, 1881: Stump nearly healed | — | The constant recurrence of suppuration round the knee necessitated amputation. |
| Left knee swollen, tibia slightly rotated out and displaced back. Synovial membrane pulpy. Articular ends red and covered with flocculent synovial membrane. Caseating centres in both condyles of femur | Wears leather splint. Small sinus unhealed | Jan. 1, 1885: "Limb strong and sound, but slightly flexed. No shortening" | — |
| Synovial membrane pulpy, ends of bones hyperæmic. Cartilages eroded. Caseous spot in femur | Patient went out wearing a Martin's bandage and a leather splint. ¾" shortening, wound healed. Slight enlargement of inguinal glands | Jan. 1, 1885: Leg slightly flexed. 1 inch shortened; bones firmly united, but boy is very delicate, and has a little pain in leg | — |

| No. | Initials, sex, and age. | Disease. | Duration of disease before excision. | Previous treatment. | Admission and discharge. | Duration of stay in hospital. | Operations performed. |
|---|---|---|---|---|---|---|---|
| 92 | E. W., F., 5 | Pulpy, right knee | — | — | A. April 21, 1881<br>D. Sept. 11, 1881 | 4 months | Excision June 11, 1881 |
| 93 | A. R., F., 12 | Pulpy, right knee | Injury 5 years ago | Extension and rest | A. June 29, 1882<br>D. Sept. 26, 1882 | 3 months | Excision July 11, 1882 |
| 94 | B. K., M., 7 | Pulpy, right knee | Injury to knee 15 mos. ago | Plaster-of-Paris splint | A. May 31, 1882<br>D. Oct. 5, 1882 | 4 months | Excision June 6, 1882. July 10, 1882: Abscess opened. Aug. 29, 1882: Numerous sinuses slit up and scraped |
| 95 | W. W., M., 25 | Pulpy, right knee | 6 mos. | Rest and strapping | A. Oct. 18, 1882<br>D. Nov. 3, 1882 Died | 16 days | Excision Oct. 27, 1882 |

| State of joint at time of excision. | State on discharge. | After history. | Remarks. |
|---|---|---|---|
| Leg cannot be extended within 60°. Tibia dislocated back and rotated out, joint is swollen, no pain. Bones healthy. Cartilages eroded. Synovial membrane pulpy | Patient went out with plaster - of - Paris splint. No shortening. Union not quite firm, allowing slight movement | Nov. 29, 1881: Right leg slightly flexed. 1″ shortening. Dec. 16, 1881: Readmitted for leather splint to be applied, with which he went home | — |
| Leg flexed at angle of 140°. Knee swollen. Synovial membrane pulpy. Cartilages eroded on femur and patella. Caseous osteitis in inner condyle of femur | Patient went out with wound almost healed, and bones united in good position | — | — |
| Knee swollen; leg flexed at angle of 135°. Tibia displaced back and rotated out. Synovial membrane pulpy; cartilages eroded | Oct. 5, 1882: Patient developed scarlatina and was sent away with bones united but some sinuses unhealed | April, 1883: Wound healed completely at Scarlet Fever Hospital, leg strong and straight. Went to school and soon left off splint. | A large quantity of pulpy granulations developed round knee, and the effect of slitting up 13 sinuses and scraping them produced a most marked improvement. |
| Feb., 1884: Mother says that leg has only bent during last month and has become painful. Plaster-of-Paris splint applied to prevent increased flexion.<br>March 6, 1884: Two sinuses have formed recently on inner side of knee. The leg is flexed 30°. Extension applied.<br>June 16, 1884: Two sinuses formed on outer side of knee.<br>Aug. 22, 1884: Sinuses slit up. $HNO_3$ applied.<br>Sept. 28, 1884: Superficial sinus on outer side of knee nearly healed; leg nearly straight.<br>Jan. 1, 1885: "Leg straight, sound, and strong. No shortening (?)" | | | |
| Knee swollen, flexed. Movement very limited. Synovial membrane pulpy. Cartilages eroded. Caseating and sclerosed ostitis of femur | P. M. — Strumous right kidney. A few chronic tubercles in right lung. Tubercular intestine. During life no albumen or pus in urine | — | Left thigh amputated at lower ⅓rd when 8 years old for pulpy knee. Having only one leg, he was very desirous of its being saved. After the excision he was sick and sleepless. Temperature rose to 106°, and he died in 7 days. Viscera found extensively diseased. |

| No. | Initials, sex, and age. | Disease. | Duration of disease before excision. | Previous treatment. | Admission and discharge. | Duration of stay in hospital. | Operations performed. |
|---|---|---|---|---|---|---|---|
| 96 | E. S., F., 21 | Pulpy, left knee painful and swollen. Worse 10 months. Joint has been suppurating and discharging caseous pus for 2 months | 6 yrs. ago knee | Rest | A. Dec. 20, 1881<br>D. Apr. 6, 1882 | 3½ months | Excision Dec. 31, 1881 |
| 97 | E. J., F., 11 | Pulpy, left knee. Partial ankylosis with displacement and flexion | Kick on knee 6 mos. ago | Rest, extension with weights | A. Nov. 24, 1881<br>D. Apr. 1, 1882 | 4 months | Excision Jan. 3, 1882 |
| 98 | O. P., M., 41 | Partial ankylosis of left knee-joint femur running into knee-joint. Worse 3 months | 10 mos. ago fracture of left | Rest and splints | A. Mar. 11, 1882<br>D. July 13, 1882 | 4 months | Excision Mar. 24, 1882. May 12, 1882: Sinuses slit up. June 4, 1882: Abscess on post. surface of leg opened |
| 99 | F. B., F., 11½ | Inflammation of right knee arising from disease of tibia | 3 mos. ago inflammation in left leg with necrosis of tibia | Rest, removal of sequestrum from tibia | A. Jan. 25, 1879<br>D. Sept. 22, 1879 | 8 months | Mar. 13, 1879: Sequestrotomy, right tibia. June 26, 1879: Excision, right knee |
| 100 | E. W., M., 7½ | Pulpy, right knee | 2½ yrs. ago measles. Knee inflamed soon after | Splints and rest for 2 years | A. Apr. 9, 1879<br>D. Dec. 15, 1879 | 8 months | April 10, 1879: Excision |

| State of joint at time of excision. | State on discharge. | After history. | Remarks. |
|---|---|---|---|
| Knee swollen, painful, and suppurating. Sinus on outer side. Synovial membrane pulpy. Cartilages destroyed, both surfaces of bone undergoing caseous osteitis with large sequestrum in each | Patient left wearing a Croft. Incision healed. 3″ shortening | Jan. 1, 1885: Leg straight, strong, and sound. A little shortening<br>Dec. 16, 1888: Limb straight and strong, but very little shortening. Is in good health | The osteitis extended deeply into both bones so that large pieces were sawn off and the ends gouged out.<br>— |
| Knee flexed, cannot be extended beyond 135°. 1″ shortening of tibia. ½″ shortening of femur. Caseating osteitis of head of tibia. Cartilage was covered with fibrous tissue, and was wasted | Leg slightly bowed when patient left. Walks with crutches | — | |
| Impaired movement. Patella ankylosed to callus of T-shaped fracture into joint. The cartilages had almost entirely disappeared | Went out, leg straight and strong, using crutches | April, 1884: Has been to sea again. Leg strong and straight<br>Oct., 1888: Leg perfectly straight and strong | A Norwegian sailor, in whom firm ankylosis took place within six weeks. The suppuration afterwards was entirely superficial, and was due to the great rapidity with which the skin united before the deep parts had completely closed. |
| Right knee-joint painful and swollen; limited movement | Wound healed. Leg straight. Wearing a leather splint | July 17, 1880: Union firm. 1½″ shortening. Knee slightly bent in and foot inverted. Wearing leather splint.<br>Oct. 28, 1880: Superficial abscesses round *left* knee which do not communicate with joint | Seen about 2 years later. A useful limb. |
| Leg flexed at right angle to thigh, swollen. All the bone surfaces were eroded | Wearing leather splint. Small granulating surface remaining still | Jan. 1, 1885: "Leg sound, strong, and straight; some shortening" | A small popliteal abscess was opened on Jan. 21st, 1880, and the sinus remained open for nearly four months, but finally healed. |

| No. | Initials, sex, and age. | Disease. | Duration of disease before excision. | Previous treatment. | Admission and discharge. | Duration of stay in hospital. | Operations performed. |
|---|---|---|---|---|---|---|---|
| 101 | A. I., F., 12 | Pulpy, left knee | 1 year | Splints and counter-irritants | A. May 12, 1879<br>D. Dec. 20, 1879 | 7 months | Excision June 2, 1879. Sinuses slit up Sept. 11, 1879 |
| 102 | A. Y., F., 11 | Pulpy, left knee | 7 years | Cold affusions, rest, and bandaging | A. June 16, 1879<br>D. Sept. 22, 1879 | 3 months | Excision July 7, 1879 |
| 103 | E. J., M., 7 | Pulpy, right knee | 2 years | Blisters and strapping. Extension | A. May 17, 1880<br>D. Mar. 27, 1881 | 10 months | Excision Oct. 21, 1880 Jan. 31, 1881 Sinus slit up |
| 104 | M. W., M., 5½ | Pulpy, right knee<br>injured knee. Abscess in popliteal space | 21 mos. ago in- | Poultice and rest | A. Jan. 22, 1881<br>D. Sept. 14, 1881 | 8 months | Excision Feb. 4, 1881. Mar. 17, 1881: Abscess on inner side of knee opened. June 30, 1881: Sinuses slit up |
| 105 | A. P., F., 5 | Pulpy, right knee | 3 years | Plaster-of-Paris splint | A. Feb. 17, 1881<br>Death<br>D. May 26, 1881 | 3 months | Excision Mar. 3, 1881 |
| 106 | K. D., F., 5 | Pulpy, left knee | Within last year measles, scarlatina, and pertussis. Knee swollen 6 mos. | McIntyre splint. 1 month | A. Oct. 29, 1881<br>D. Mar. 23, 1882 | 5 months | Excision Dec. 1, 1881. Sinuses scraped Jan. 19, 1882. Abscess over head of fibula Feb. 9, 1882. Amputation through lower ⅓ femur Feb. 15, 1882 |

| State of joint at time of excision. | State on discharge. | After history. | Remarks. |
|---|---|---|---|
| Sinus on outer side of knee, which is flexed, and movement is much impaired | Leg straight. Wearing leather splint. Spinal abscess is again discharging | Died in April, 1881, from the spinal caries; knee was consolidated during the acuteness of the knee affection, but broke out again after the consolidation of the knee. | In 1877 had spinal caries, with abscess, for which she wore a Sayre's jacket. The back became quiescent |
| Knee swollen. Movement impaired. Synovial membrane pulpy. Cartilages eroded | Wound quite healed | Jan. 1, 1885: "Limb sound, straight, and strong. No shortening." | — |
| Right knee is flexed and swollen. Sinuses round joint. Tibia dislocated backwards. Caseous osteitis in head of tibia. Synovial membrane pulpy | Wound healed. Wearing splint. July 16, 1881: Abscess opened over inner condyle of femur. Carious bone felt. Aug. 30, 1881: Sinuses well. Splint left off. | Jan. 1, 1885: Leg sound and strong, bowed outwards at the knee. 1 inch shortening. The splint was left off much too soon | — |
| Right knee enlarged, semiflexed. 2 sinuses on outer side of joint. Caseous osteitis at end of femur. The abscess sac extended some distance beneath quadriceps | Wound has almost entirely healed. | Jan. 1, 1885: Limb straight, strong, and sound. About 1 inch shortening | Epiphysial cartilage partly removed on outer side of femur on account of osteitis. July 4, 1881: Scarlatina with nephritis later on. |
| Knee flexed and swollen. Caseating osteitis of inner condyle of femur. Hard gummatous tumour on synovial membrane | — | — | 2 sisters died of phthisis. May 5, 1881: Wound healed; union firm between bones. May 15, 1881: Developed tubercular meningitis, of which she died on 25th. Union between bones quite firm (in R. C. S. Mus.). |
| Synovial membrane pulpy. Feb. 15, 1882: Bones only united at one part. Position not good. Quantity of caseating pulpy material between and around bones | Stump healed | Died May 29, 1883. Causes unknown | Child in very feeble health at time of excision. |

| No. | Initials, sex, and age. | Disease. | Duration of disease before excision. | Previous treatment. | Admission and discharge. | Duration of stay in hospital. | Operations performed. |
|---|---|---|---|---|---|---|---|
| 107 | N. V., F., 6 | Pulpy, right knee | 6 mos. ago mor- | Back-splint and strapping | A. Feb. 1, 1882<br>D. Apr. 12, 1882 | 2 months | Excision Feb. 23, 1882 |
| | | billi, pertussis and scarlatina; knee swollen since | | | | | |
| 108 | M. C., M., 4½ | Pulpy, left knee | 3 years | Extension. Plaster-of-Paris splint | A. July 23, 1882<br>D. Oct. 10, 1883 | 15 months | Oct. 5, 1882. Excision<br>Feb. 15, 1883: Sinuses slit up |
| 109 | W. H., M., 7 | Pulpy, left knee | 1 year | — | A. Oct. 16, 1882<br>D. Mar. 10, 1883 | 5 months | Excision Oct. 26, 1882 |
| 110 | A. B., M., 9 | Pulpy, knee | Knee swollen 6 years | Splints and rest. Extension with weights. Knee-joint kept in a vapour bath | A. Nov. 20, 1882<br>D. Mar. 30, 1883 | 3 months | Excision Feb. 8, 1883 |
| 111 | G. B., F., 9 | Pulpy, right knee | 4 years | Splints. Elastic cap | A. Oct. 31, 1883<br>D. Feb. 15, 1884 | 4 months | Excision Nov. 8, 1883 |
| 112 | M. R., F., 11 | Pulpy, right thigh and knee. Caseating cervical glands, with sinuses | 7 mos. | Rest. Splints | A. May 20, 1881<br>D. Mar. 1, 1885 | — | July 7, 1881: Superficial abscess over knee opened<br>Sept. 29, 1881: Excision of knee<br>March 16, 1882: Sinuses slit up |

| State of joint at time of excision. | State on discharge. | After history. | Remarks. |
|---|---|---|---|
| Knee flexed and swollen. Synovial membrane pulpy. Patella eroded | Wound quite healed | Jan. 1, 1885: Limb sound, strong, and straight; no shortening" | March 1, 1882: Scarlatina. Wound continued to heal well. |
| Knee flexed at angle of 150°; tibia partially displaced backwards. Caseous ostitis of tibia with sequestrum. Synovial membrane pulpy | Wearing leather splint. Small sinus still open. Leg is in good condition. | Feb. 15, 1883: No bony union; pulpy material between ends in places<br>Jan. 1, 1885: Leg sound and strong; flexed at an angle of 45°; considerable shortening. The splint had been left off much too soon | Oct. 21, 1882: Scarlatina. Wound continued to heal well. Strumous, unhealthy child. |
| Knee flexed; slight movement. Synovial membrane pulpy. Cartilages eroded on outer side of joint. Bones healthy | Wearing leather splint. Wound not quite healed | May 3, 1883: Wound healed.<br>Jan. 1, 1885: "Limb sound, straight, and strong; about 1 inch shortening" | — |
| Knee flexed. Cartilage eroded. Caseous ostitis on outer head of tibia. Synovial membrane pulpy. Outer parts of cartilages of tibia and femur removed | Leg straight and strong. Wound healed. | Jan. 1, 1885: Limb straight, sound, and strong; no perceptible shortening | — |
| Knee flexed; tibia dislocated back, and rotated outwards. Cartilages eroded, and the granulation had extended into outer condyle of femur | Wearing leather splint. Leg firm and straight; no shortening apparent. | Jan. 1, 1885: Limb strong, slightly flexed, 1¼" shortening; still a small sinus | — |
| — | — | — | Commenced as pulpy infiltration of cellular tissue about knee; pus formed over patella and under quadriceps. Joint at first unaffected. Periosteum became secondarily implicated; afterwards the knee-joint. Friends persistently refused amputation. After the excision union between the bones (fibrous) was firm in March, 1882, but thigh and popliteal space were riddled with sinuses. Lardaceous disease of liver, spleen, and kidney set in. During 1883-84 the sinuses, both in neck and thigh, slowly healed, and during 1885 the lardaceous affection lessened, and when the child left hospital she was much better. |

| No. | Initials, sex, and age. | Disease. | Duration of disease before excision. | Previous treatment. | Admission and discharge. | Duration of stay in hospital. | Operations performed. |
|---|---|---|---|---|---|---|---|
| 113 | A. K., F., 19 | Permanent flexion of right knee after infantile paralysis | 17 years ago infantile paralysis | Extension was applied when she was 4 years old. No treatment since | A. Mar. 25, 1881<br>D. Mar. 1, 1882 | 11 months | Excision April 23, 1881 |
| 114 | A. K., F., 19 | Permanent flexion of left knee after infantile paralysis | Do. | Vide supra | A. June 7, 1882<br>D. Sept. 27, 1882 | 3 months | Excision June 13, 1882 |
| 115 | A. W., F., 19 | Disease of right knee secondary to necrosis of part of femur | Necrosis of lower end of right femur 7 years ago | Sequestra removed at intervals. Jan. 30, 1883: One protruding above knee was removed. $4\frac{1}{2}'' \times 1''$ wound partially granulated up | A. June 20, 1883<br>D. Oct. 17, 1883 | 4 months | June 27, 1883 Excision |
| 116 | F. J., M., 6 | Abscess opening into right knee-joint | Injury to right thigh one week before admission; large abscess formed in lower part between muscles of thigh and communicated with knee-joint Oct. 3, 1882: Opened | — | A. Sept. 23, 1882<br>D. Mar. 25, 1883 | 6 months<br>16 weeks after excision | Dec. 1, 1882: Excision |

| State of joint at time of excision. | State on discharge. | After history. | Remarks. |
|---|---|---|---|
| Both knees flexed. Right tibia displaced back, and rotated out. Double talipes equino-varus. Legs of equal length; right knee 1½″ larger circumference. She generally crawled on this knee. Partial movement in right knee; thigh everted; right leg warmer than left. 2½″ removed from ends of femur and tibia. There was no evidence of any disease. Cartilage thin, with vessels running over the surface | Wound quite healed. Wearing leather splint. Leg strong and straight | Jan. 1, 1885: Limb sound<br>May 6, 1881: Patch of skin over calf gangrenous; toes cold; loss of sensation, due probably to stretching of the nerves.<br>June 8, 1881: Slough on heel.<br>Sept. 27, 1881: Erysipelas of thigh.<br>Nov. 13, 1881: Redness of leg with bullæ. | The right leg has, as long as patient can remember, been drawn up, with heel close to buttock. The left leg is powerless, and has only lately been drawn up. She can only get about by crawling on hands and knees. |
| Movement much impaired in left knee. Thigh inverted. Tibia displaced backwards and rotated outwards. Foot cold and in an inverted position. Joint tissues healthy. Cartilage thin. Sufficient bone removed to get limb straight | Wound healed | June 4, 1883: Small toe, left foot, amputated for onychia and malposition | This patient suffered various troubles after both the excisions, from the very imperfect state of the circulation in the limb. She gradually improved, and in 1885 came up to the hospital, and walked with sticks about the ward, to show how much better she was. |
| Knee flexed; thigh wasted; movement limited. Tibia displaced back and rotated out. 1¼″ of end of femur removed, extremely soft and fatty. Overgrowths of bone on surfaces of condyles, a mass of fibroid inflammatory material ¾″ thick, round patella | Wound healed, leg straight and strong. 4½″ shortening | — | — |
| Patella attached to condyles by dense fibrous tissue which extended also over external condyle of femur. Bones and remaining synovial membrane and cartilage healthy | Wound healed, leg straight | — | — |

| No. | Initials, sex, and age. | Disease. | Duration of disease before excision. | Previous treatment. | Admission and discharge. | Duration of stay in hospital. | Operations performed. |
|---|---|---|---|---|---|---|---|
| 117 | A. N., F., 3 | Pulpy, right knee | Injury 1 year ago | Rest. Extension | A. Feb. 8, 1882<br>D. Feb. 12, 1883 | 1 year 11 months after excision | Excision March 21, 1882 |
| 118 | E.F.G., F., 3½ | Pulpy, right knee | Pain and swelling 14 mos. Suppuration of joint 11 mos. | Splints. Extension. Rest 9 months | A. Jan. 13, 1883<br>D. July 15, 1883 | 6 months 4 months after excision | Excision March 20, 188 |
| 119 | J. D., M., 39 | Pulpy, left knee | Injury 2½ years ago. 1 year ago injured knee again | Blisters. Splints. Elastic stocking | A. Nov. 20, 1882<br>Died June 26, 1883 | 6 months | Excision Jan. 5, 1883. Abscesses opened Feb. 21, 1883. March 2, 1883 Amputation through thigh |
| 120 | G. W. H., M., 17 | Pulpy, left knee | Injury 2 years ago, and again 4 mos. ago | Rest. Counter-irritants, treated by bone-setter for "dislocated patella." Reduction under $CHCl_3$ massage | A. Mar. 24, 1883<br>D. Nov. 15, 1883 | 8 months | Mar. 24, 1883 Hæmatoma over patella aspirated, ʒij of blood drawn off<br>May 18, 1883: Excision.<br>July 27, 1883: Sinuses slit up.<br>Oct. 9, 1883: Amputation through thigh |
| 121 | W. B., M., 10 | Pulpy, left knee | 1 year ago injury, and also 6 mos. ago | Rest. | A. Nov. 7, 1882<br>D. Feb. 28, 1883 | 3½ months | Excision Nov. 17, 1882 |

| State of joint at time of excision. | State on discharge. | After history. | Remarks. |
|---|---|---|---|
| Knee enlarged, flexed; impaired movement. Small amount of pus in joint. Cartilages eroded. Synovial membrane pulpy | Leg strong and straight. Two small chronic sinuses, one on either side of limb | — | Abundant suppuration about excised ends of bones for a long time with sinuses in popliteal space.<br>Oct. 31, 1882: Erysipelas.<br>Nov. 20, 1882: Leg straight, union good. Still unhealed sinuses. |
| Joint swollen. Knee flexed, movement limited. Sinuses round knee leading to synovial pulpy sac of joint. Cartilages covered with dense fibroid material | Leg straight, bony union. Sinus on either side | Sept. 12, 1883: Sinus still discharged, wearing leather splint.<br>Jan. 1, 1885: Limb straight, sound, and strong. No shortening | — |
| Knee swollen, flexed; cartilage of femur eroded. Sequestrum with caseous osteitis in head of tibia. Synovial membrane pulpy. | — | — | Jan. 26, 1883: Cough; aphonia. Abundant, muco-purulent expectoration.<br>Feb. 26, 1883: Chronic phthisis, chiefly right lung.<br>March 2, 1883: Extensive suppuration between muscles round excised joint. Thrombus in post-tibial vein. Bony surfaces firmly united by fibroid material except at post. and outer part where there was abscess cavity<br>April 25, 1883: Patient about ward on crutches.<br>June 26, 1883: Died with general tuberculosis of lungs with large cavities at apices. |
| Knee flexed, swollen. Synovial membrane pulpy 1¼″ thick. Caseous osteitis of inner condyle of femur from pulpy fringe. Patella very dense, no trace of fibro-cartilages. | Stump healed, except some small granulations | — | — |
| Oct. 9, 1883: No bony union between ends; anterior part covered with granulations and fibrous tissue; posterior part bare and looked as if recently sawn. No necrosis. Suppuration among muscles. No caseous material | | | |
| Knee swollen, flexed. Synovial membrane pulpy. Caseous osteitis of inner condyle of femur | Union firm, legs straight, wearing leather splint | April 11, 1883: Leg strong and straight.<br>Jan. 1, 1885: "Limb strong and sound, slightly flexed. ⅞″ shortening" | Mother syphilitic.<br>Jan. 5, 1883: Bony union, leg straight, *i.e.* 7 weeks after operation. |

| No. | Initials, sex, and age. | Disease. | Duration of disease before excision. | Previous treatment. | Admission and discharge. | Duration of stay in hospital. | Operations performed. |
|---|---|---|---|---|---|---|---|
| 122 | J. S., M., 32 | Chronic disease of right knee | Gonorrhœal rheumatism of right knee 10 yrs., 6 yrs., and 1 month ago | — | A. Feb. 8, 1883<br>D. May 29, 1883 | 3½ months | Excision Feb. 16, 1883 |
| 123 | C. W., M., 21 | Pulpy, left knee, with articular necrosis | Injury 5 yrs. ago, gradually getting worse | Splint. Scott's ointment | A. Nov. 9, 1882<br>D. Oct. 7, 1883 | 11 months | Excision Nov. 24, 1882. Dec. 13, 1882: Sinuses slit up. April 24, 1883: Amputation through lower ⅓ thigh. May 23, 1883: Free incisions on outer side of thigh. Sloughing fascia removed |
| 124 | M. K., F., 38 | Pulpy, left knee, with articular necrosis of both bones | Knee inflamed 5 yrs. ago, and has been worse for 5 mos. | Blisters, splint | A. Oct. 9, 1882<br>D. May 10, 1883 | 7 months | Excision Oct. 17, 1882. Sinuses slit up Jan. 11, 1883 |
| 125 | A. C., F., 44 | Pulpy, right knee, with articular necrosis of both bones | Injury 7 yrs. ago. 5 yrs. ago leg became flexed, painful, and swollen | Splints. In 1880 under a bone-setter, leg straightened under ether. Ice poultices. Counter-irritants | A. April 7, 1883<br>D. Aug. 12, 1883 | 4 months | Apr. 10, 1883: Excision. June 13, 1883: Sinuses slit up |

| State of joint at time of excision. | State on discharge. | After history. | Remarks. |
|---|---|---|---|
| Movement fair, lower end of femur thickened. Synovial membrane pulpy. Cartilage on external condyle of femur eroded. Bones healthy | 1″ shortening; leg straight, union firm, small granulating surface on outer side; wearing leather splint | Jan. 1, 1885: "Limb straight, strong, and sound. About ½ inch shortening" | Node over sternum. Perforation of posterior palate. Urethritis, stricture. |
| Knee swollen. Movement limited. Tibia partially dislocated outwards and backwards. Synovial membrane pulpy, eroding outer condyle of femur. Ends of bones indurated, caseous in spots, and excavated by granulations. | Oct. 7, 1883: Went to country for a change. Extensive burrowing on outer side of thigh. Liver much enlarged. Stump not completely healed | — | There was free suppuration for months after the excision, and but little bony union took place. After 5 mos. the leg was amputated. |
| April 24, 1883: Large abscess cavities in popliteal space, leading to a necrosed sheath of bone 4″ long over posterior and lower surfaces of femur with considerable periosteal osteophytic growth over shaft of femur generally | | | |
| Joint enlarged; movement impaired. Cartilages had for the most part disappeared. Sequestrum $1\frac{1}{2} \times 3\frac{1}{4} \times 1''$ in cavity in head of tibia, and 2 sequestra in femur. A small one in outer condyle, and one $\frac{3}{4} \times 1 \times \frac{3}{4}''$ in inner condyle | May 10, 1883: Leg is quite healed. Union firm, and leg straight. Wearing leather splint | Jan. 1, 1885: Limb straight, strong, and sound. Two inches shortening<br>Jan., 1889: Limb remains in the same favorable state | Jan. 11, 1883: Union firm. Leg straight. |
| Knee flexed, swollen; movement impaired. Abscess cavity extended 7″ under rectus muscle. Ends of both bones much eroded, with caseous and eburnated osteitis. Sequestrum in head of tibia $1\frac{1}{2} \times \frac{5}{8} \times 1$ inches | Aug. 12, 1883: Two small sinuses with slight discharge. Leg strong and straight. Wearing splint | May, 1884: Leg sound and useful.<br>Jan. 1, 1885: Limb sound, straight, and strong. Two inches shortening | June 13, 1883: Good bony union. |

| No. | Initials, sex, and age. | Disease. | Duration of disease before excision. | Previous treatment. | Admission and discharge. | Duration of stay in hospital. | Operations performed. |
|---|---|---|---|---|---|---|---|
| 126 | W. P., M., 13 | Pulpy, right knee | 5 years | Rest, splints | A. Sept. 22, 1883<br>D. Mar. 3, 1884 | 5 months | Oct. 16, 1883: Excision |
| 127 | W. H., M., 23 | Multiple articular necrosis of ends of bone; secondary to pulpy disease | Injury 6 and 4 years ago to left knee; flexed and useless 2 years | Rest. Splints. Counter-irritants | A. Oct. 17, 1883<br>D. Feb. 5, 1884 | 3½ months | Excision Nov. 2, 1883 |
| 128 | F. B., F., 8 | Pulpy, left knee | 3 years | Rest. Splints. Iodine | A. Dec. 22, 1883<br>D. April 29, 1884 | 4 months | Excision Jan. 25, 1884 |
| 129 | H. H., M., 3 | Pulpy, right knee | Injury 4 mos. ago | Scott's ointment | A. June 25, 1884<br>D. Oct. 10, 1884 | 2½ months | Excision July 18, 1884 |

*Note.*—The 130th excision is Case No. 51, in which both knees were excised.

| State of joint at time of excision. | State on discharge. | After history. | Remarks. |
|---|---|---|---|
| Knee flexed and swollen. Tibia rotated out and dislocated back. Synovial membrane pulpy, and eroding the ends of both tibia and fibula. Bone fatty | Mar. 3, 1884: Leg quite strong and straight. Wound well. Wearing leather splint | Jan. 1, 1885: Limb sound, straight, and strong. No shortening (?)<br>Dec. 17, 1889: Limb strong, but slightly bent; thinks about 2″ shortened. (Letter) | Feb. 2, 1884: Severe attack of tonsillitis lasting 2 weeks. |
| Joint flexed, painful; movement limited. tibia dislocated backwards, and rotated out. Cartilages had almost entirely disappeared. Head of tibia much eroded; a sequestrum $\frac{1}{2} \times \frac{1}{2} \times \frac{1}{4}$″ lay in cavity; rest of bone denser than normal. End of femur much and deeply eroded. Necrosed bone $\frac{3}{4} \times \frac{1}{2} \times \frac{1}{2}$″, but attached at upper end, and several nodules of loose dense bone, $\frac{1}{2}$″ diameter | Feb. 3, 1884: Union firm; wound healed; leg straight. Wearing leather splint | Jan. 1, 1885: "Limb sound, straight, and strong. 1″ shortening" | — |
| Knee flexed and swollen. Synovial membrane pulpy. Bones healthy | April 29, 1884: Leg straight; union good; wound healed. No apparent shortening. Wearing leather splint | Nov. 2, 1884: Very slight flexion of bones. Leg strong and painless.<br>Jan. 1, 1885: Limb strong and sound, and but very slightly (8°) flexed. No shortening | Died at home Aug. 22, 1885. Cause unknown, but apparently unconnected with knee, which was sound and strong. |
| Knee swollen, painful; flexed movement limited. Tibia rotated outwards. Synovial membrane pulpy in places. Caseating pus in joint. Cartilage eroded in places. Bones healthy | Sept. 10, 1884: Wound not healed. Bones united. Leg straight. Small granulating wound | Jan. 1, 1885: Limb sound and strong; slightly flexed; no shortening. Pain in popliteal space when he stands on leg. Thickening of lower end of femur; several small abscesses have been opened. Utterly filthy and neglected at home | The child was taken away to Yarmouth before the wound had healed, as the parents were moving. The bones had united firmly. |

TABLE II.—*Showing Duration of Malady before Excision in each Case.*

| | |
|---|---|
| From 10—20 years . | . Cases Nos. 1, 13, 51, 63, 66, 69, 113, 114=8 cases. |
| From 5—10 years . | . Cases Nos. 2, 4, 7, 11, 14, 22, 32, 37, 40, 46, 58, 65, 72, 85, 86, 93, 96, 102, 110, 115, 122, 123, 124, 125, 126, 127=26 cases. |
| From 2—5 years . | . Cases Nos. 3, 5, 9, 10, 12, 18, 19, 23, 24, 27, 28, 29, 31, 34, 35, 38, 42, 43, 47, 54, 62, 67, 71, 74, 75, 76, 77, 79, 80, 89, 90, 91, 100, 103, 105, 108, 111, 119, 120, 128=40 cases. |
| From 1—2 years . | . Cases Nos. 6, 15, 20, 25, 30, 36, 39, 41, 44, 53, 56, 57, 61, 70, 73, 81, 83, 94, 101, 104, 109, 117, 118, 121=24 cases. |
| From 6 months to 1 year | . Cases Nos. 8, 33, 45, 49, 52, 55, 60, 78, 82, 87, 88, 95, 97, 98, 106, 107=16 cases. |
| Less than 6 months . | . Cases Nos. 16, 17, 21, 26, 48, 50, 59, 64, 84, 99, 112, 116, 129=13 cases. |

In Cases Nos. 68 and 92 the duration of the disease is not reported on.

# PERITONEAL SANGUINEOUS CYSTS

AND THEIR RELATION TO

# CYSTS OF THE PANCREAS.

By THEODORE FISHER, M.D.

(Communicated by the Editors.)

The blood-cysts that it is intended to consider in this paper constitute a well-defined class. They are situated partially or entirely between the layers of the peritoneum, are non-malignant in nature, and are not secondary to inflammation of the peritoneum.

Pelvic hæmatocele, the large sanguineous cysts sometimes associated with soft sarcomata, and the smaller effusions occasionally met with in subacute peritonitis have not therefore been included.

Three theses have appeared in Paris dealing with this variety of abdominal tumour:—Chayron, 1877; Rouiller, 1885; and Bérard, 1888. The last, that by Bérard, entitled "Des hématomes du Mésentère," deals mainly with effusions in the layers of the mesentery associated with strangulated hernia; but the second, that by Rouiller ("Les kystes hématiques du Péritoine"), is an interesting and valuable contribution to the subject.

The following is a brief outline of the clinical features of a case:

A patient, who may have received a blow on the abdomen, suffers from symptoms (sometimes called dyspeptic) of varying severity, most commonly taking the form of abdominal pain associated with vomiting and constipation. After a period of uncertain length a tumour is discovered. This is aspirated, dark brown fluid containing blood-cells coming away. The patient generally completely recovers.

It will be noted that there is a resemblance in this clinical history to that of pancreatic cysts. I shall endeavour at the end of this paper to show that the relation of the two classes of cyst is probably very close. Brief notes of two cases of hæmorrhage between the layers of the peritoneum have been introduced amongst the cases of sanguineous cysts, and for the sake of convenience have been numbered continuously with them.

The following case came under my own observation, and I have to thank Dr. Gross, of Westbourne Grove, late Medical Superintendent of the St. Saviour's Union Infirmary, East Dulwich Grove, for permission to publish it.

Case 1.—H. B—, a cab driver, æt. 59, was admitted into the Infirmary March 18th, 1891.

He had suffered from rheumatic fever when eleven years of age, and eight years before admission he had had abscesses in the right arm, which, it was said at St. Bartholomew's, were due to glanders. While recovering he had a second attack of rheumatic fever. Six months before the present admission he had been in the Infirmary for "pain in the chest and heart." There was then apparently nothing very much the matter with him; a soft systolic apex bruit, however, was noted.

He gave a history of having got wet while driving his cab on two successive snowy days—March 9th and 10th. On March 15th he had severe pain in the lower part of the abdomen, followed by vomiting. The vomiting became very severe, and for three days he had kept nothing down. The bowels had not been opened since the 12th, six days before admission. He had not previously had any similar attack.

*Notes on admission.*—Face flushed, breathing rather hurried, and chiefly thoracic. Abdomen very tender, the slightest touch makes him call out ; the recti abdominis are slightly rigid, but the other muscles are not unless pressed upon. Right iliac region rather more tender than left, no rigidity nor fulness. Has no abdominal look. Nothing abnormal in lungs. Soft systolic bruit best heard in fifth space, half an inch outside nipple line. Urine, sp. gr. 1030, deposit of urates, considerable quantity of albumen. Temp. 100·6°.

The patient was rather thin, and had a somewhat distressed appearance, but did not look acutely ill, and although something was supposed to be the matter with him, one could hardly help thinking that much of his trouble was subjective.

The acute symptoms of pain and vomiting lessened in two or three days, the latter becoming infrequent. Constipation continued, the bowels being opened at three or four days' interval.

The next notes are on March 30th, twelve days after admission. Temperature has been gradually rising the last few days. Vomited bilious fluid three days ago, but not since ; complained of pain in the epigastrium on 28th. This morning the upper part of abdomen is swollen and tender. Lower border of swelling situated just below umbilicus, and curves upwards to ribs on either side. The tenderness does not allow of much examination. Swelling slightly more prominent to left of middle line ; dull on percussion. Abdomen supple below swelling, but still slightly tender, especially towards the left groin.

As noted above, the temperature was 100·6° on admission. On the third day, as the symptoms abated, it fell to normal, where it remained for five days, when it again began to rise, and reached its highest point, 101·6°, on the 29th, the day before the above notes were taken. Nine days afterwards it had again become normal.

The swelling appeared somewhat suddenly. Before the 28th the epigastrium was rather sunken than distended ; but on that date, when pain in that region was first complained of, the patient said that he noticed fulness commencing.

April 9th.—He vomited a large quantity of dark blood,

estimated by the nurse at four pints, without the abdominal swelling being in any way affected.

On the 10th a small patch of discoloration, like a bruise, was noticed just below the umbilicus.

On the 13th the notes are—

Vomited three times yesterday, bringing up 11 oz. of blood, and 2 oz. again in the night. Blood vomited yesterday bright in colour, but on the 9th it was more or less coffee-ground.

The tumour remains about the same size, is most prominent just above and to the left of the umbilicus, is flattened and ill-defined towards the epigastrium, with rounded curved border below; surface smooth, slightly tender, more towards the lower border on the left side. Dull to percussion, but a narrow strip of resonance separates it from the liver and from the left edge of ribs. Pulsates slightly, but it is difficult

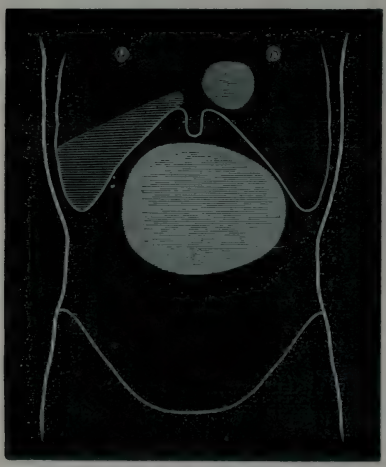


FIG. 1.—Diagram showing position and extent of dulness on April 13th.

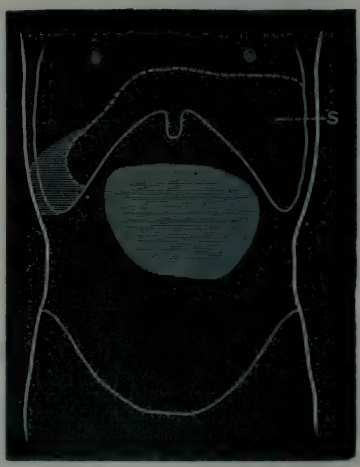



FIG. 2.—Diagram showing area of stomach resonance (s) April 20th.

to say whether the pulsation is communicated from the aorta or heart.

16th.—Passed about 3 oz. of blood *per rectum*; said by the nurse to have been a clot dark in colour, but easily recognised as blood.

20th.—Stomach resonance very well marked, as high as

the fifth space on the left side; easily obtainable in seventh space in nipple line through the liver dulness on the right. Tumour still dull and slightly tender. The lower border stretches downwards from the tenth rib on either side. Does not move with respiration. Is elastic and resistant and a thrill is obtainable. Patient has been improving lately. Complains of little pain. Has not vomited for over a week. Complexion a yellowish sallow colour; conjunctivæ white.

23rd.—Over the most prominent part of the swelling, about two inches to the left of the umbilicus, Dr. Gross inserted an exploring needle, and drew off some dark

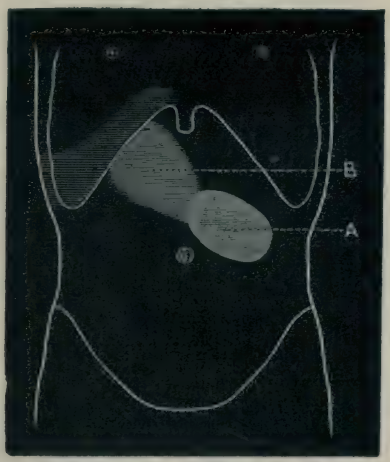



FIG. 3.—Diagram showing small tumour that remained after tapping. A. Prominent area, showing size of tumour after aspiration. B. Ill-defined dulness stretching obliquely upwards to that of liver.

brown fluid, the colour of strong thick coffee. The fluid spurted out through the needle after removing the syringe, and was allowed to drain away. About 40 oz. were evacuated. Under the microscope the deposit of the fluid showed abundant brownish-coloured granules and numerous deformed red blood-cells.

On the following day the notes are—The swelling much smaller and altered in shape, the area of dulness having completely changed. The hemispherical shape has now entirely disappeared. The only prominent part of the tumour is just above and to the left of the umbilicus. It is

smooth, rounded, and tender. An area of impaired resonance is continuous with the liver dulness.

On May 2nd the swelling was less, but there was still some tenderness. Early in June, however, all trace had disappeared, and the man went out shortly afterwards perfectly well.

Eighteen months after the man was again seen. He was stouter and looked well, and was most emphatic when speaking of the improvement in his health since his illness, and declared that he could eat "a beef-steak easily," which apparently he could not do before. Like most cabmen, however, he, without doubt, was given to taking rather too much alcohol, and complained of a "nasty heaving" in the morning.

The systolic bruit, formerly audible, could not be heard, but the first sound was somewhat prolonged.

The urine was high-coloured, sp. gr. 1026, acid, contained no albumen and no sugar.

Inquiries were made for some abdominal injury as a possible cause of the abdominal cyst. He stated that about two months before admission he had been thrown over the top of his hansom cab, striking the abdomen just below the umbilicus, against the metal rest for the reins. Bruising of the lower half of the abdomen followed.

He apparently, however, had not thought of connecting this injury with the sudden onset of the illness for which he was admitted to the infirmary, since it followed closely upon getting wet on a cold day.

In the above case a tumour, which proved afterwards to be a cyst containing blood, appeared within the course of forty-eight hours in the upper part of the abdomen. It was aspirated successfully, and eighteen months after the patient was in good health. Later we shall notice several similar cases, but before looking at these it will be interesting to consider a few points in connection with the situation of the above tumour.

It was a well-defined cyst lying mainly in front of the intestines in the upper part of the abdomen. The rapidity with which it attained a size that it did not afterwards exceed at once suggested the pouring out of fluid into some pre-existing cavity. This cavity was thought at the time to

be that contained between the layers of the great omentum, and afterwards it was considered possible that effusion originating in the upper part of the lesser peritoneal cavity had travelled downwards between these layers.

It may be necessary to remind some of our readers that the lesser cavity of the peritoneum does not stop short at

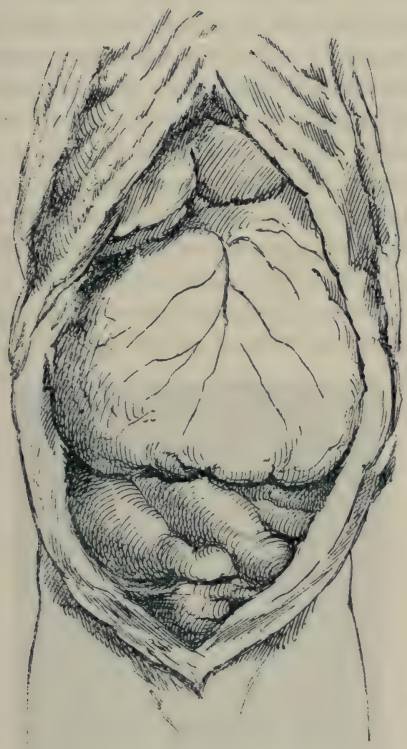


FIG. 4.—Sketch of the great omentum, distended with plaster of Paris, in a child aged eleven months. The liver lies above; below are the small intestines and bladder.

the greater curvature of the stomach, nor at the upper surface of the transverse colon, but extends downwards between the layers of the great omentum. Since this is the case, the cavity varies in extent with the length of this portion of the peritoneum. The tumour we are considering only

descended to a point just below the umbilicus, while the great omentum most commonly extends lower, and may reach to the pubes. This fact seemed to militate against the idea that the effusion was in this situation.

It was decided to endeavour to inject the cavity, in order to see what form a collection of fluid in it would be likely to assume.

On November 12th, 1892, a paper appeared in the 'British Medical Journal,' by Mr. Jordan Lloyd, on "Injury to the Pancreas a cause of effusions into the lesser Peritoneal Cavity." He had made injections in the dead subject, using plaster of Paris, which was also employed to inject the lesser peritoneal cavity of the child, from which the diagram (Fig. 4) is taken. The omentum in this case was very short, and was curled up just below the transverse colon. On being unravelled, it extended about one third of an inch below the umbilicus. The method of injection was as follows:

A portion of the great omentum was nipped up just below the greater curvature of the stomach, the catheter

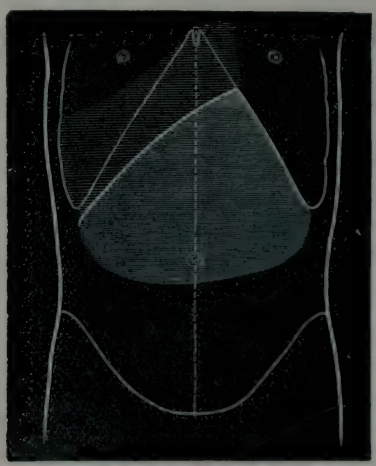


FIG. 5.—Diagram showing area of dulness over great omentum distended with plaster of Paris after the abdominal wall had been sewn up. Along the inner half of the white line, marking the edge of the liver, percussion had to be very light to avoid bringing out stomach resonance.

passed back into the concavity to the left of the spinal column, and tied in. Plaster of Paris was then injected.

It filled the hollow in which the end of the catheter lay, travelled down the left side of the cavity of the great omentum, then rising over the front of the spine, escaped by the foramen of Winslow into the general peritoneal cavity, at the same time that it filled the right and middle portions of the cavity of the omentum.

The central incision in the abdominal wall having been sewn up, the area of dulness produced by the distended cavity of the omentum was mapped out. It will be seen in Fig. 5 that this dulness reached to just below the umbilicus. Reference to Figs. 1 and 2 will show that the dulness, though less extensive, extended downwards to exactly the same point. This is striking, but, as will be shown afterwards, is not conclusive. It is possible that some cysts in this region may be effusions into the lesser peritoneal cavity; but there is strong evidence to show that cysts in this region are not, as a rule, in this anatomical position. Before examining that evidence, however, we will glance at the cases published by Mr. Jordan Lloyd in the 'British Medical Journal,' November 12th, 1892, as examples of effusion into the lesser peritoneal cavity following injury to the pancreas.

A man, æt. 20, was admitted into the General Hospital, Birmingham, in August, 1888, three months after an injury. "A tumour could be seen and felt in the epigastrium and left hypochondrium, tense and rounded, reaching from the left costal arch down to the umbilicus, and into the upper part of the left lumbar region. It was dull on percussion and tender to pressure." Twenty-nine ounces of fluid were drawn off by aspiration. The cyst filling up again laparotomy was performed and the cyst opened. The patient died, and at the post-mortem the following is the description:

"Situated behind the stomach, and co-extensive with its posterior surface, was an enormous encysted hæmatoma, its lower edge reaching further than the stomach, and to within one and a half inches of the transverse umbilical line. It was removable with the stomach, and was filled with three or four pints of dark blood containing old coagula; thus the comparatively empty and collapsed stomach appeared to

rest posteriorly upon a large sac (the lesser peritoneal cavity), which was distended with altered blood."

Mr. Jordan Lloyd seems to have thought the situation in this case to have been unquestionable, but the pathologist states that, "regarding the structures entering into the composition of the sac walls, the folds of the lesser omentum were so altered that it was hardly possible to tell what they were, but it appeared that hæmorrhage might have occurred from some ruptured vein in the gastro-splenic omentum." It must be remembered that the lesser peritoneal cavity is not a hollow space, but that under ordinary circumstances it is filled up by the apposition of the various structures around it. Unless the layers of peritoneum can be traced around the walls of the cyst, it would be impossible to state definitely the position of the effusion. It was in this case admitted that even such a well-defined structure as the lesser omentum was scarcely discernible, having been altered by the extravasation of blood. The want of definition of these boundaries, and the fact that the cyst was "removable with the stomach," would lead to the acceptance of Mr. Lloyd's conclusion with some reserve. The next case is similar, but did not die.

A man, æt. 27, also with a history of injury, was admitted into the Birmingham Workhouse Infirmary, December 9th, 1891.

Fig. 6, reproduced by the kind permission of Mr. Jordan Lloyd, shows the position of the tumour in this case. It is described as "a large protruding swelling occupying nearly the whole umbilical, the lower part of the epigastric, and the left hypochondriac regions, tense on palpation, fairly well defined, dull on percussion, the dulness not continuous with that of the liver or spleen. A resonant area can be percussed out all round the swelling. There is a dull area in the back, extending from the level of the angle of the scapula to the lowest ribs, where the breath sounds and vocal fremitus are diminished but not absent."

On December 12th the swelling was aspirated and 20 oz. of dark-brown turbid fluid drawn off. It was rich in albu-

men, and deposited "a thick layer consisting of blood-cells, débris of cells, and linear-like crystalline bodies."

On the 18th laparotomy was performed. The following is Mr. Jordan Lloyd's description of the operation:—"I opened the abdomen by an incision 2½ inches long in the middle line above the umbilicus. The cavity of the peritoneum was quite healthy. The greater curvature of the stomach lay across the upper end of the opening, with a red, thickened, great omentum passing downwards from it; the omental vessels were abnormally large and tortuous, and were engorged with blood, the vein being as thick as a

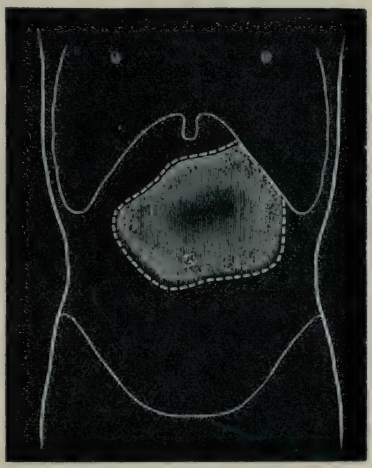


FIG. 6.—Position of tumour in one of Mr. Jordan Lloyd's cases.

penholder. A fine needle was pushed through the omentum and fluid found; the omentum was then stretched carefully to the skin, the parietal peritoneum not being included in the sutures; an opening was then made through the omentum, and 84 oz. of brownish turbid fluid were caught in receivers. A finger passed into the opening entered a cavity, situated behind the stomach, in front of the pancreas, above the colon below the left lobe of the liver, limited to the right, but passing deeply backwards to the left, beyond reach of the finger."

The position of the cyst in this case seems still less conclusive than in the one that precedes it. The fact

that it apparently had the shape of the lesser peritoneal cavity does not prove that the wall of the cyst was formed by the boundaries of that cavity.

In the second case the fluid removed at the operation contained blood, but was also found to emulsify fats and to possess amylolytic properties. This led Mr. Jordan Lloyd to suggest that some of the reported cases of pancreatic cyst have really been effusions into the lesser peritoneal cavity due to injury of the pancreas, the escaping blood setting up inflammation.

Although apparently digressing, since the question has some bearing upon our subject, we will briefly examine a few of the reported cases of pancreatic cyst, in order to see whether these cysts are commonly situated in the lesser peritoneal cavity. It will be found that it is mentioned, either that the great omentum lies free in front of the cyst, or that the transverse colon lines its lower border. Were the fluid under tension in the lesser peritoneal cavity it would, of course, invade the cavity of the omentum, the layers of which would be taken up into the wall of the cyst, and press the transverse colon backwards, so that it would lie behind instead of below.

The first case was under the care of Dr. Newton Pitt, and was operated upon by Mr. Jacobson. By kind permission of Dr. Pitt a diagram showing the position of the tumour has been reproduced from a paper read before the Medico-Chirurgical Society, June 9th, 1891.

A glance at Fig. 7 will show that the dulness in this case was more extensive, and descended lower than in Mr. Jordan Lloyd's. At the time of the operation it was apparently smaller, but the cyst evidently at least equalled Mr. Lloyd's in size.

At the operation the stomach was found tightly stretched over the subjacent cyst, and the omentum lay free lower down in front of it. Mr. Jacobson found considerable difficulty in dealing with this portion of the peritoneum, and finally had to remove a considerable portion of it before he could attack the cyst. Here, as in both of Mr. Lloyd's cases, there was effusion behind the stomach, this organ being tightly stretched over it, yet the cavity of the great

omentum was not invaded. Evidently, in this case, at least, the contained fluid was not free in the lesser cavity of the peritoneum.

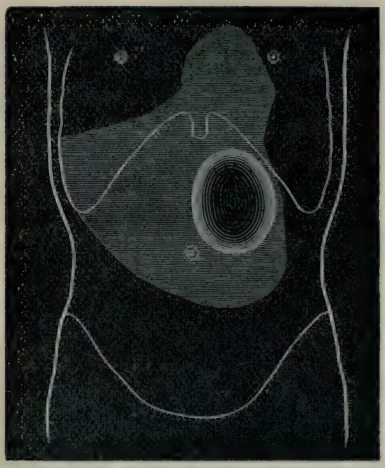


FIG. 7.—Diagram showing area of dulness in case of pancreatic cyst under the care of Dr. Pitt.

Examination of other cases quoted by Dr. Senn seems to point to the same conclusion.

In Riedel's case,[1] although the tumour filled the whole abdomen at the operation, the "slightly adherent omentum was easily separated," and the "transverse colon was seen immediately behind the symphysis pubis." This last point suggests that the cyst was between the layers of the transverse mesocolon, and that the large intestine had been carried downwards. Had the fluid been in the lesser cavity of the peritoneum the transverse colon would have been pressed backwards, since the fluid, when entering the prolongation of that cavity between the layers of the omentum, would pass in front of that portion of the large intestine.[2] In Lardy's case (quoted by Dr. Pitt) the transverse colon was stretched tightly over the cyst, while in Rokitansky's an attempt to remove the cyst caused a rent

[1] 'International Journal of Medical Sciences,' 1886 and 1887.

[2] In this case the mesocolon is mentioned, but its relation to the cyst is not clear; apparently it was thought that it had been severed from its posterior attachment.

in the large bowel. In Salzer's case the transverse colon lined the lower border of the cyst, and when the intestine was elevated it is definitely stated that it was found that "the cyst belonged to the mesocolon."

That not only pancreatic cysts, but that hæmorrhagic effusions may lie between the layers of the mesocolon, is shown by the following interesting case brought before the Medical Society, December 5th, 1881, by Dr. Isambard Owen.

Case 2.—A woman, æt. 57, was brought to St. George's Hospital in a condition of collapse. She died an hour and a half later.

At the post-mortem "a black mass was seen occupying the position of the liver, but extending lower down into the abdomen. It was found to be a mass of recent blood-clot enclosed between the layers of the transverse mesocolon, and extending a little way into the great omentum. It occupied all the upper portion of the abdomen, pushing up the stomach, liver, and diaphragm."

Nothing could be discovered in the blood-vessels that threw any light upon the source of hæmorrhage. All the abdominal organs were healthy. The heart muscle was flabby and thin, and the fatty coating in excess. The mitral valves were thickened at their edges, and one aortic valve was thickened. The lungs showed a few tuberculous nodules. There was no evidence of embolism nor of syphilis. Dr. C. T. Williams mentioned that he had also seen a case of hæmorrhage into the transverse mesocolon in a woman who died of tuberculosis. He thought the hæmorrhage might have been secondary to vomiting.

In Dr. Owen's case it is worthy of note that, although the hæmorrhage was between the layers of mesocolon, it distended those layers sufficiently to displace the liver and diaphragm upwards. Mr. Jordan Lloyd laid stress upon dulness at the base of the left side of the chest as diagnostic of effusion into the lesser peritoneal cavity. If, however, the diaphragm can be displaced upwards by hæmorrhage into the transverse mesocolon, an effusion in that situation would probably produce the same physical signs.

To recapitulate the various points considered in connection with the anatomical position of cysts in the upper part of the abdomen. It has been seen that artificial distension of the lesser peritoneal cavity may, when the omentum is short, produce a tumour resembling in shape and size the cysts that occur in that region. Examination, however, of the evidence afforded by the cases brought forward by Mr. Jordan Lloyd to support the view that some cysts in the neighbourhood of the pancreas are collections of fluid in the lesser peritoneal cavity, shows that it fails to be conclusive; while reported cases of pancreated cyst seem to point to the transverse mesocolon as the more common site.

The case of Dr. Isambard Owen, quoted above, also lends strong support to the latter view.

In the light of the above facts we may with some reason conclude that the first case mentioned in this paper was a case of hæmorrhage into the transverse mesocolon, not, however, of quite so acute a nature as that of Dr. Owen's, and consequently not terminating fatally.

Sanguineous cysts of the abdomen may occur in other regions of the abdomen; but before noticing them we will look at a few brief notes of cases of cysts in a similar position to those just described.

The following case is recorded by Dr. Clifford Allbutt in the 'Lancet,' 1883, vol. ii, p. 13.

Case 3.—A stout man, æt. 30, with an abdominal tumour, was admitted into the Leeds Infirmary. He gave the following history:—Four years previously he had suffered from an attack of dyspepsia, which passed off, but five months before admission it recurred, and was accompanied by vomiting. He then noticed a hard lump, the size of a closed fist, in the epigastric region, which was said to have disappeared a few days after the symptoms had subsided. Between that time and admission he had three similar attacks, all being accompanied by reappearance of the tumour, which had failed to disappear since the last attack, a fortnight before admission. He had been temperate. There was no history of syphilis.

*On admission* there was constant vomiting with pain. A

distinct rounded tumour could be felt occupying the whole of the epigastric region, extending downwards to the umbilicus, and laterally beneath the ribs, inclining rather to the left of the median line. It was elastic and freely moveable from side to side; it did not appear to be attached to either the liver or spleen. There was no ascites and no cardiac nor pulmonary lesion. The urine was high coloured and contained mucus. He remained in the hospital three weeks and then went out, the tumour being smaller. Readmitted four months after, the tumour was much larger, and extended a couple of inches below the umbilicus. An exploring syringe was introduced, and fluid drawn off containing broken-down blood-cells, granules of colouring matter, and some large and more perfect nucleated cells of epithelial type. The tumour was afterwards aspirated, and 32 oz. of fluid withdrawn. A month afterwards, however, the tumour had regained its former size. It was again aspirated, and 51 oz. drawn off. The right side, however, diminished much more than the left, and another aspiration was performed in a different spot, 32 oz. being withdrawn. Everything disappeared, and he went out ten days after.

He was seen as an out-patient, and the tumour was slowly reappearing when, three months after, he had an attack of vomiting and diarrhœa, the motions being black. The tumour disappeared, and when the patient was seen at the end of another two months there was nothing to be felt.

In this, as in the first case, aspiration in one spot did not completely empty the cyst. This case of Dr. Allbutt's elicited a letter from Dr. Menzies, of Galashiels, giving the following particulars of a case seen by him, which had terminated in a similar way ('Lancet,' 1883, vol. ii, p. 348).

Case 4.—Man, æt. 20, when seen complained of severe intermittent pain in the abdomen, growing worse at night, and sometimes becoming intense. Vomiting occasionally occurred. This went on for about six weeks, and he became weak and emaciated; about this time an abdominal tumour was noticed, which rapidly increased in size. It was situated between the umbilicus and the epigastrium,

and soon attained the size of a fœtal head, having a globular form and a feeling of elasticity; when the tumour began to assume this size the pain somewhat abated. The tumour was aspirated, and a few ounces of reddish-brown liquid drawn off, but an accident to the aspirator prevented the operation being completed. Diarrhœa came on three or four days after; the stools consisted of greenish-black viscid matter, a considerable quantity coming away. The tumour completely disappeared. Eight years after he was in perfect health.

Another very similar case is recorded by Dr. Sinclair, of Dundee ('Lancet,' 1885, vol. i, p. 614).

Case 5.—A man, with a history of intemperance, æt. 51, was admitted to the Dundee Royal Infirmary. Two months before he had noticed a round swelling just below the umbilicus. On admission a rounded swelling, quite smooth to the touch, could be made out in the centre of the abdomen. It occupied the umbilical part of the epigastric, hypochondriac, and right lumbar regions, and a very small portion of the hypogastric. It was separable from the liver by a narrow resonant line, and it did not move with respiration. Hepatic and splenic dulness were normal. There were no abnormal signs in other organs. The appetite was good, the bowels constipated. Urine pale, 1010, acid, no albumen. The cyst was aspirated, and 60 oz. of fluid withdrawn, containing abundant deposit of red blood-corpuscles. The tumour had quite disappeared on the following day. He went out a fortnight later.

Seen a year after, Dr. Sinclair thought he detected some signs of reaccumulation, but the man was doing his usual work.

In the following case the swelling was in a similar position to those described above, and proved to be a cyst containing fluid that deposited red blood-cells. It is recorded in the 'Lancet,' December 10th, 1892, by Dr. Sutherland, as a case of "Traumatic Non-suppurative Peritonitis."

Case 6.—A girl, æt. 8, was admitted into the Paddington Green Children's Hospital on August 8th, 1888. She had been knocked down by a cab, the wheel of which was said

to have passed over her abdomen. She complained of great pain in the back and in the epigastrium, and lay in bed with her thighs flexed on the abdomen.

On the following day the abdomen was distended and tender in the epigastric region. She suffered greatly from thirst and was sick after taking food. The breathing was entirely thoracic, the muscles of the abdomen being kept rigid. Three weeks later pain and sickness had ceased, but the temperature at night ranged between 100° and 101°. In the epigastric region a distinct swelling could be seen and felt, extending downwards from the ensiform cartilage to within an inch of the umbilicus, and passing beyond the edge of the rectus muscle on each side. The tumour was aspirated a fortnight later, and ten ounces of dark green fluid removed. The fluid contained a large amount of albumen and red blood-corpuscles. She was discharged well in three weeks, but there still remained some thickening in the epigastric region.

It is obvious that, with the exception of the case of Dr. Izambard Owen (No. 2), all these cases bear a great resemblance to one another. In all the five cases there is a tumour of almost identical size and shape in the upper part of the abdomen, which was proved to be a cyst containing a dark-coloured fluid. In four cases this fluid was examined microscopically and found to contain red blood-cells. In three, withdrawal of the fluid effected a cure, in the other two the contents escaped by the bowel. In three abdominal pain and vomiting preceded or accompanied the appearance of the cyst.

The singular appearance and disappearance noticed early in Dr. Clifford Allbutt's case, we shall see has occurred in others.

In the last case (No. 6) there was a definite history of traumatism. When looking at the reports of sanguineous cysts in other parts of the abdomen we shall find that several followed an injury, sometimes quickly, sometimes not until several weeks had elapsed.

It may be thought by some readers that all the preceding cases have merely been examples of pancreatic cyst, in which the fluid that was withdrawn was not tested for amylolytic action or other distinctive features. Before arriving at that

conclusion we will look at a few similar cysts in other parts of the abdomen.

In the following three cases a cyst containing either sanguineous or sero-sanguineous fluid was connected with the mesentery.

Case 7 (M. Terrier, 'Société de Chirurgie,' 1890, p. 383).—A man, æt. 34, since the end of October, 1889, had suffered from attacks of colic from time to time. They lasted four or five hours, ending with the passing of a normal motion or with diarrhœa. On March 16th, 1890, he was taken with colic more severe than usual. He kept his bed on the 17th, the abdomen being somewhat swollen. On the 19th he was admitted to l'Hôpital de la Charité. The colic continued, with constant pain in the hypogastrium, disturbing sleep. On April 2nd a puncture was made and 450 grammes of blood-stained fluid withdrawn, followed by abatement of symptoms.

The bowels were regular. The urine contained no albumen and no sugar. The cyst refilled, and was then described as a rounded cyst reaching from the pubes to the umbilicus, and laterally to within two fingers' breadths of the iliac spines on either side. It was somewhat mobile. At an operation on May 12th, a tumour the size of a fœtal head at term was exposed, apparently connected with the mesentery. It is stated that "above the tumour adhered to the small intestine, to the right it adhered to the mesentery, to the left it was easy to separate two omental adhesions, which were tied with silk and resected." The wall of the cyst was some millimetres in thickness, and the internal surface was found coated with fibrin coloured with blood. The contents appeared to be apparently pure blood. The cyst was removed and the patient recovered. On May 21st he was said to be considered well.

The resemblance of this case to many of those in which the cyst was situated in the upper part of the abdomen is at once apparent. A history of dyspeptic attacks, with the appearance of a tumour during one of them, is common to nearly all that have not quickly followed an injury.

In the following case, unfortunately, no history is given, but it is interesting from the fact that the blood effusion is

distinctly stated to have been situated between the layers of the mesentery.

Case 8 (M. Potherat, 'Bulletins de la Société anat.,' 1891, p. 202).—Woman, age not given. A fluctuating spherical tumour was situated in the umbilical region of the abdomen, but invaded both the epigastric and hypogastric regions. It was everywhere surrounded by a zone of resonance, and was unconnected with the liver or the pelvic organs. It was thought to be a hydatid cyst. At the operation it was found free from adhesions, lying between the layers of the mesentery.

When punctured a liquid described as "ocreuse" in colour escaped. On opening the cyst more fully it was found to contain a large quantity of solid friable material of the same colour. The wall of the cyst was lined unequally by stratified layers of blood-clot, some of which were decolourised. The woman died, but no full account of the post-mortem examination is given.

Another case of sero-sanguineous cyst of the mesentery is reported by Frentzel in a monograph on mesenteric cysts in the 'Deutsche Zeitsch. für Chirurgerie,' 1892. The following is taken from an abridged translation in 'La Semaine médicale,' May 25th, 1892.

Case 9.—A woman, æt. 20, was admitted under Lücke with a history of slow increase in size of the abdomen, constipation, and some wasting. On the right side of the abdomen was a rounded fluctuating tumour about the size of a child's head, reaching as high as the right hypochondrium, and, below, a few fingers' breadths below the umbilicus. It was very mobile, and, under chloroform, could be pushed across the middle line into the left hypochondrium. It was attached neither to the liver nor to the organs of the pelvis. An exploratory puncture drew off sero-sanguineous fluid.

At the operation the cyst was found to lie between the layers of the mesentery. The cyst was fixed to the abdominal wall and opened three days later. The wound healed entirely in some months.

The above is a sero-sanguineous cyst, but a case we shall

afterwards quote of blood effusion connected with the pancreas seems to show that such cysts may lose their distinctly sanguineous character. Possibly some serous cysts of the mesentery may have commenced as a blood effusion.[1] It is, however, remarkable, as we shall see later, how nothing but blood, or blood and blood-clot, may be found in a cyst that has been known to have existed for years.

We cannot leave the mesentery without quoting an extremely interesting case of death from hæmorrhage into its layers, reported by Dr. Gilbart Smith at the same meeting of the Medical Society as the case of death from hæmorrhage into the transverse mesocolon already referred to.

Case 10.—A carman, æt. 25, was suddenly seized with abdominal pain while drinking a glass of beer.

When admitted to the London Hospital a few hours after, he was suffering from severe abdominal pain and repeated vomiting. He died seventeen and a half hours from the onset.

At the post-mortem the small intestines were found to be of deep purple colour, and there was a large extravasation of blood between the layers of the mesentery, more especially into that portion connected with the lower three feet of the ileum. The peritoneal surface was everywhere shining, and presented neither a milky appearance nor signs of inflammatory lymph. A small inguinal hernia, about three inches long, was found about two feet from the cæcum, and in its neighbourhood the bowels were intensely discoloured. Extravasation of blood into the muscular coat of the bowel at the neck of the diverticulum was especially noted. The mucous membrane of the intestines was highly congested, and studded with numerous patches of blood extravasation. The colon, stomach, and upper part of the intestines were congested, but in a much less degree. The contents of the intestines were mainly liquid, but the colon contained formed fæces.

[1] At a meeting of the Clinical Society, May 13th, 1887, Mr. Godlee related three cases of abdominal cyst following injury. Apparently blood was not present, but two of them, in other details, closely resemble the cysts we are considering.

No aneurism, constricting band, nor volvulus was found. The heart and other organs were healthy.

Dr. Gilbart Smith mentioned that the act of drinking suggested irritant poisoning, but the condition of the mucous membrane negatived this view. He stated that he was unable to give an explanation of the hæmorrhage. There was no lesion elsewhere that might have given rise to embolus.

This case is somewhat complicated by the presence of the hernia. It was, however, very small, and evidently was not considered to have anything to do with the effusion of blood. It may be mentioned that M. Bérard, in his thesis, gives notes of effusions into the mesentery of limited area in six cases of strangulated hernia, and seems to think that they throw light upon the pathology of peritoneal sanguineous cysts. He states that hæmorrhages associated with herniæ are due to a mechanical lesion of vessels; and, with regard to the sanguineous cysts, adopts the view of Dr. Williams, that they may be secondary to vomiting. M. Bérard, however, does not appear to be very familiar with the reported cases of these cysts, at least he only refers to the cases mentioned by the various observers at the meeting of the Medical Society of London in 1881.

The conditions of a portion of the mesentery nipped in one of the abdominal rings are so obviously different from those of the various folds of peritoneum free in the abdominal cavity, that the possibility of a similar causation for hæmorrhage in the two positions is not worth serious consideration. To return to the above case, one can hardly think that a hernia 3 inches long can have led to a blood effusion into the mesentery connected with 3 feet of intestine, unless partial strangulation had led to thrombosis of a mesenteric vessel which spread upwards for a considerale distance. No thrombosis was, however, discovered. I should prefer to look upon this case as an instance of a rare but curious liability of the layers of peritoneum to hæmorrhage.

Having glanced at the mesentery, we will pass on to a few cases of blood-cyst in other parts of the abdomen. The following case is an example of a cyst in the great omentum. Previously I have suggested that in some in-

stances a sanguineous effusion may sooner or later become sero-sanguineous; in this case, however, there was a history of a tumour having been present for twelve years, and yet the contents at the time of death were mainly blood and blood-clot.

Case 11 (M. Simon, 'Bulletins de la Société anatomique,' 1858, p. 30).—Man, æt. 44, admitted to l'Hôpital Saint Antoine for Asiatic cholera.

He was thought to have a distended bladder, since there was a rounded tumour in the lower part of the abdomen, stretching upwards from the hypogastrium to the umbilicus, but extending rather to the right of the median line. So convinced were the residents that it was a distended bladder that he was repeatedly catheterised. He was almost unconscious on admission, but afterwards he improved slightly, and they were able to elicit the fact that the tumour had existed for nearly twelve years, but that it had gradually increased in size.

He died, and at the post-mortem the tumour was found to be a cyst of the great omentum, adherent above to the great curvature of the stomach. The walls were fibrous, rather thin, and the contents granular coagulated blood, the colour of rust. At the upper part of the cyst was a greyish friable mass, described as like brain-substance macerated in alcohol for some time. This substance was furrowed by channels and diverticula containing liquid blood. The gastro-epiploic arteries and veins were of normal size, and did not seem to be in any way altered. All the rest of the organs of the body were healthy, except the large intestine, in which there were a few small follicular ulcers.

When noticing the cases of sanguineous effusion between the layers of the mesentery, it is possible that the thought may have occurred to the reader that the blood had travelled downwards from the vicinity of the pancreas, and in the above case a similar origin may have suggested itself. Here, however, the cyst did not reach up so high and far back as the pancreas, apparently stopping short at the great curvature of the stomach. The effusion, however,

need not have been in the cavity of the omentum. It may have been, and in all probability was, between either the anterior or posterior layers of the omental fold. In connection with this a glance at a diagram of a case under Dr. Bantock, published in the 'Transactions of the Obstetrical Society' for 1881, by Mr. Alban Doran, would be of interest. That diagram, if referred to, will be seen to show very clearly how a cyst can exist in the great omentum without invading the upper and hinder part of the lesser peritoneal cavity. The transverse colon, and with it the mesocolon, is pressed upwards, and lies in contact with the stomach, thus completely shutting off the upper part of the lesser peritoneal cavity. In this case there is no definite evidence that the cyst was sanguineous, although the repeated withdrawal by tapping of a "dark serous fluid" suggests such an origin. It was present in a woman aged fifty-eight, and had existed for several years.

On leaving the great omentum and looking at the neighbourhood of the small omentum, we shall find collections of blood closely applied to the pancreas, but without having apparently any definite connection with the gland.

Case 12 (M. Richet, 'L'Union médicale,' 1877, vol. xliv, p. 82).—A man æt. 42, on July 15th, 1876, was knocked down by an omnibus and was taken to l'Hôpital St. Louis. Three false ribs were bruised on the left side and one on the right. He was a healthy man and had never been ill. After being in the hospital six weeks he went out, but a pain remained in his side, which prevented him from resuming his former occupation, which consisted in carrying heavy weights throughout the day. His health, however, continued good. He had no dyspeptic trouble.

On January 10th, 1877, he discovered a tumour on the left side of the abdomen.

He was admitted to l'Hôtel Dieu February 19th, 1877. The tumour is then described as situated under the diaphragm, projecting behind the abdominal wall to the left of the linea alba; it was quite round, fluctuating, and painless, and did not exert pressure on surrounding organs. There was no dyspeptic disturbance. It was punctured, and

1500 grammes of a red clear liquid containing albumen obtained. It deposited red blood-cells but no hooklets, and gave no reactions for bile constituents.

On March 15th the tumour had reappeared, and was larger than before. It was decided to inject the cyst with a weak cauterising fluid. The adoption of this method of treatment caused fatal peritonitis.

At the autopsy the lesser omentum was seen to be occupied by a cyst, which had stripped the peritoneum for some little distance from the posterior wall of the stomach. Although adherent to the liver it was seen to have no pathological connection with it. It is described as being limited above by the liver, below by the pancreas, and in front chiefly by the small omentum, but partly by the stomach. All the surrounding organs, the pancreas, spleen, and kidneys, were healthy, and normal in situation. The cyst-wall was about 3 mm. in thickness, the internal surface furrowed and flocculent and covered with ecchymoses. Microscopically it was seen to be composed of connective tissue with numbers of flat cells.

As mentioned before, although the cyst touched the pancreas, it appears to have had no other connection with it than that of mere apposition. The pancreas is distinctly stated to have been healthy, and there were probably two layers of peritoneum separating the gland from the cyst. The situation of the cyst in the following case appears to be the small omentum.

Case 13 (M. Rendu, 'Bull. Soc. anat.,' 1880, p. 120).—Man æt. 38. He enjoyed good health until October, 1875, when he was suddenly seized with colic and bilious vomiting in the middle of the night. He was taken to l'Hôpital St. Antoine, where he complained of pain in the left side of the abdomen, with vomiting and constipation. A tumour, supposed to be an enlarged spleen, was present. He went out in January,[1] 1876. In July vomiting reappeared and lasted six months, but there was no return of pain.

In May, 1879, vomiting again returned after each meal.

[1] The size of the supposed enlarged spleen is not then mentioned, but in June, 1879, it is said to be of normal size.

He went to l'Hôtel Dieu, where he was said to have dilated stomach. He went out in June, and after being out a few days entered l'Hôpital Tenon. He was emaciated, and had the cachexia of a patient suffering from cancer. The stomach descended to the umbilicus.

On October 3rd the right lobe of the liver was thought to be hypertrophied.

On October 20th there was a tumour in the same situation, about the size of the palm of the hand, projecting slightly, but difficult to define. It was tense but fluctuating. It was thought to be connected with the right lobe of the liver. It was punctured, and a yellowish grey fluid drawn off containing globules of pus, albumen, some mucin, bile colouring-matter, and phosphates, but no hooklets.

November 25th.—The cyst had refilled and was again punctured, a similar fluid being drawn off.

December 16th.—A third puncture. This time the fluid was of chocolate colour; on standing it separated into two layers, the upper dark brown, and a lower containing mucus and pus.

January 7th.—A fourth puncture, and a dark liquid drawn off.

February 22nd, 1880.—The patient died of peritonitis, following the attempted introduction of a drainage-tube through a cannula, and after washing out with weak carbolic lotion. Instead of the cyst, the fluid had entered the peritoneal cavity.

At the post-mortem there was a cyst, the situation of which is not very accurately defined. Speaking of the liver, it is stated that it was in contact below with a cavity, the walls of which were somewhat thick and resistant. This cavity was situated immediately above the stomach, of which it seemed an integral part. The tissues of the lesser peritoneal cavity were thickened as far as the pancreas and the semilunar ganglia. The stomach was dilated and hypertrophied, the pylorus being compressed by the tumour. On cutting the wall near the pylorus, a small cavity the size of a hazel nut was found situated below the mucous membrane, which communicated with the cystic cavity behind by a tortuous course, through which a pen could be easily passed.

Microscopically, the wall of the cyst was seen to be composed of fibrous tissue and embryonic cells.

The communication with the wall of the stomach is of some interest. Probably the tension of the cyst had forced its contents through a weak spot. In the succeeding case a somewhat similar complication was present, but, in addition, communication was established with the peritoneal cavity, the escape of the contents setting up a fatal peritonitis.

Mucus and mucin are said to have been present in the fluid drawn off. The correctness of that observation must be doubtful. Since, too, the cyst was found not to communicate with the liver, the colouring matter mentioned must have been derived from effused blood, and was not due to the presence of bile.

In the following case the cyst is apparently situated mainly between the muscular and peritoneal coats of the posterior wall of the stomach. The fact that in Case 10 it is mentioned that the blood effusion had stripped the peritoneum off a portion of this wall renders the correctness of this view probable.

Case 14 (M. Gullois, 'Bulletins de la Société anat.,' 1889, p. 556).—Domestic servant, æt. 22.

In August, 1883, she had pains in the left side of the abdomen, following constipation of four days' duration. This pain, sometimes associated with constipation and vomiting, lasted in a slighter degree until March, 1884, when she noticed a tumour the size of a hen's egg in the left hypochondrium.

She was admitted to l'Hôpital Tenon, March 10th, 1884. There was still pain, but no mention is made of vomiting.

The tumour, which had considerably increased in size, was tapped four weeks after admission. A clear, highly albuminous fluid containing no hooklets was drawn off. The tumour and the pain disappeared for a fortnight, when the pains returned, and by the end of another eight days the tumour had reappeared. The tapping was repeated, but in another fortnight the tumour had again reappeared. The tumour was supposed to be a cyst of the spleen, and the patient was transferred to the care of M. Lucas Champon-

nière. The tumour as described at the time appears to have been extensive. It was said to occupy the hypochondrium, flank and upper half of the iliac region on the left side, and invaded the right side as far as the junction of the median and lateral regions. Above was stomach-resonance, and below resonance separated it from the pelvis, and was also present in the lumbar region. The surface was smooth. The urine was normal.

The pain and constipation continued, but she took her discharge on June 21st.

She was readmitted August 11th, but again soon took her discharge.

Readmitted again October 22nd. Eight days before she had sudden pain in the abdomen during micturition, and vomiting returned. There were general signs of peritonitis, and she died on the following day after admission.

*Post-mortem*.—Small cavities showing signs of cicatrisation at the apices of both lungs, with tubercles throughout both upper lobes. The heart and pericardium healthy.

On opening the abdomen, the peritoneal cavity contained some sanguineous fluid, and there were numerous soft adhesions in different places, particularly between the great omentum and the abdominal wall. Liver, spleen, and pelvic organs healthy. The kidneys were said to show signs of parenchymatous nephritis.

A tumour was situated behind and below the stomach, about the size of the head of a fœtus at term, but probably larger before the escape of some of its contents into the peritoneal cavity. Its wall was well defined and rather thick. It was united to the posterior wall of the stomach and to the great omentum, but was distinctly separate from the spleen, transverse colon, and from the pancreas and the posterior abdominal wall; above it stopped some little distance from the liver. It is stated that although projecting into the lesser cavity of the peritoneum it had not its limits. In the neighbourhood of the small curvature of the stomach was a communication with the peritoneal cavity admitting the tip of the little finger.

The cyst was filled with dark blood-clot. Microscopic examination showed the fibrous wall of the cyst to be in

contact with the muscular tissue of the stomach. Recent hæmorrhage, which was thought to have originated in the wall of the cyst, had penetrated between and separated the muscular layers.

As it has been already suggested, the cyst probably lay mainly between the peritoneum and the muscular wall of the stomach. Microscopic examination showed it to be very intimately connected with the muscular layers. The interesting point is the definite statement that it did not fill the lesser peritoneal cavity, and that it was distinct from the pancreas and the posterior abdominal wall.

Since the following case did not terminate fatally the situation is of course doubtful. It is interesting as having been present in a young lady only eleven years old, and yet while occurring at so early an age there was no history of injury.

The case was mentioned to me by Dr. Goodhart, who saw the patient in consultation. The following brief notes were very kindly given me from memory by Mr. Noott, of Kensington Park Road, under whose care she was.

Case 15.—When Mr. Noott was called to see the patient, a girl æt. 11, she was complaining of pain in the abdomen, but was up and walking about, and had been out for a horse-ride that afternoon.

On examination of the abdomen a hard ridge was felt along the edge of the liver towards the epigastrium, and on the following day there was dulness and perceptible fulness on the right side of the abdomen as low as the umbilicus. The swelling increased in size for three days, and within sixty hours fluctuation became marked. There was no vomiting.

Fluid drawn off by an exploratory puncture contained red blood-cells, and large cells that Dr. Goodhart said suggested malignant disease. The case was afterwards seen by Sir Spencer Wells, and the cyst aspirated. The patient recovered without a single drawback. A year afterwards nothing could be felt. She is now, about six years after, a fine healthy young lady.

In this case there appear to have been no prodromal

symptoms, the cyst developing rapidly in an apparently healthy girl. This rapidity of development is worthy of note. The situation of the hæmorrhage was possibly the mesocolon in the neighbourhood of the hepatic flexure.

The presence of large cells is curious and interesting. Large epithelial-like cells were also noted in Dr. Clifford Allbutt's case. We are aware of their presence in peritoneal effusions, but one would hardly expect to find them associated with hæmorrhages between the layers of the peritoneum. Later on we shall find that they are common in the fluid of pancreatic cysts.

The situation of the cyst in the following case was somewhat similar. Here, post mortem, the cyst was found to be largely retro-peritoneal, and partially between the layers of peritoneum forming the hepatic flexure of the colon.

Case 16 (M. Méry, 'Bull. de la Société anat.,' 1885, p. 39). —Man æt. 76. He had noticed his abdomen to be getting larger for two years, and for six months there had been a tumour in the right hypochondrium. The tumour increased in size; pains in the hypochondrium became violent, and were associated with wasting. The bowels were constipated, but there was no loss of appetite and no vomiting.

On admission to l'Hôtel Dieu there was a smooth tumour occupying the right hypochondrium, and stretching across the epigastric region into the left hypochondrium, but not extending as low as the umbilicus. Dulness over it was continuous with liver dulness.

The tumour was aspirated, and 1½ litres of black sanguineous fluid drawn off.

The patient died after a short illness. He had some fever, with signs of pleurisy at the right base.

At the autopsy a hemispherical tumour the size of an adult head was present in the upper part of the abdomen on the right side. It had displaced the liver to the left, and had come in contact with the diaphragm. It was adherent to the lower surface of the liver, but quite distinct from it. The transverse colon was below, and the peritoneum passed from its upper surface over the anterior surface of the tumour. Behind, the tumour rested on the

right lumbar region, the kidney being below and to the left.

On opening it a great quantity of sero-sanguineous fluid escaped with a few black recent clots. The wall was lined by irregular masses of reddish-brown colour, having the characters of old blood-clot. Beneath these friable masses were organised clots; beneath these, again, however, was a black clot of recent date, and in the centre of the cyst were some similar black clots. The arteries were carefully examined, but there was no sign of aneurism. Along the back ran the inferior right diaphragmatic artery and a branch of the superior mesenteric, which were healthy. The liver was pale yellow and fatty. The heart showed no disease of the orifices, but was soft and flabby.

There was some effusion in the right pleural cavity.

Microscopic examination of the cyst-wall showed several layers of fibrous tissue, with some elastic fibres, chiefly in the inner layers. The walls contained numerous vascular canals opening into one another, and containing blood-cells.

After the patient's death it was learnt from his daughter that four years before he had received a blow on the right side from the buffer of a railway carriage.

The following case is apparently in a somewhat similar position, but is less obviously a sanguineous cyst (thesis by Rouiller, quoted by him from Chayron).

Case 17.—A carman æt. 30, received a blow from the shaft of a heavily loaded cart in the right hypochondrium. He was able to walk to l'Hôpital St. Louis, where he was admitted June 27th, 1877, but nothing was visible externally. On the following day, about 3 p.m., he had some fever, accompanied by pain in the region injured, with nausea and vomiting.

On July 4th there was distension of the abdomen, with continued vomiting and small, rapid pulse. He was thought to have peritonitis. All the symptoms, however, had disappeared by July 10th, and nothing abnormal was noticed in the abdomen. The pain in the side, however, continued. Towards the 1st of August it became more acute, and at

the same time dulness appeared in the right hypochondrium, followed soon after by fluctuation. The dulness did not alter with the position of the patient. The surface was smooth. On coughing the tumour descended *en masse*.

On August 6th the tumour was aspirated, and 2 litres of a yellow liquid, containing leucocytes and red blood-cells, were drawn off.

The tumour reappeared, and on September 25th, after washing out with tepid water, 35 to 40 grammes of pure tincture of iodine were injected. This caused considerable pain, and was followed by some symptoms of peritonitis, from which he recovered, and the injection was afterwards repeated every two days.

He left the hospital well December 12th.

The history of the above case differs somewhat from the majority, since apparently some peritonitis preceded the appearance of the tumour. It is possible, however, that this disease was merely simulated by the premonitory abdominal disturbance.

A case, apparently in the ascending mesocolon, is very briefly reported by M. Heurteaux, of Nantes, Société de Chirurgie, 1889, p. 314.

Before giving the notes he states that the cyst "was removed from its situation in the mesocolon." It contained fluid of chocolate-brown colour.

Case 18.—Woman æt. 25. The tumour was situated within the peritoneum (dans le péritoine), and displaced the cæcum and the ascending and transverse colon to the left. A strip of peritoneum was raised from over the front of it, and the cyst dissected out. The cyst was adherent to surrounding organs, especially the vena cava and liver, as well as to the broad ligament. When the case was reported the wound was healing well.

In the following case the cyst was possibly situated in a similar position on the opposite side of the abdomen.

Case 19 (M. Michalski, 'L'Union médicale,' 1877, vol. xliv, p. 400).—A healthy-looking boy, æt. 16, the son of a

farrier, was kicked, July 17th, 1876, by a horse in the upper part of the abdomen. On the following day M. Michalski was called to see him. He was suffering from epigastric pain, with repeated vomiting. There was no rise of temperature. Abdominal pain continued until September 29th, when the father of the boy called upon M. Michalski, saying that the patient had discovered a tumour in the abdomen on that same morning. When seen there was an elastic tumour on the left side of the abdomen. There was dulness from the left false ribs to about three fingers' breadth above Poupart's ligament. Dulness did not pass beyond the median line, and behind there was resonance in the left flank. The tumour was not mobile; alteration in the position of the patient had no effect upon the area of dulness. The tumour remained about the same size until November, when it somewhat increased. The dulness invaded the epigastric region, spread over the median line, and descended lower. At the same time vomiting returned and wasting set in.

December 19th.—The cyst was punctured, and 3 litres of a limpid serous fluid of the colour of light café noir came away. No mention of the discovery of red blood-cells is made. The colour seems to have been thought sufficiently diagnostic.

The patient was much relieved by the tapping. The cyst, however, filled up again, and was again tapped February 20th, 1877, the fluid being lighter in colour. On April 17th it was again tapped, and on this occasion, after evacuation, some weak liquor iodi was injected. No bad symptoms followed, and the boy was perfectly well by July 21st.

In the above case an injury is followed by a period of abdominal pain and vomiting lasting thirteen weeks before the tumour appeared. Unfortunately no mention is made of microscopic examination of the evacuated liquid; the colour, which was described as light café noir, is, however, sufficiently distinctive. That description gives a good idea of the appearance of the liquid of Case 1, which contained red blood-cells in great abundance.

Case 20 (Dr. Bristowe, 'Lancet,' 1883, vol. i, p. 163).—A

young man æt. 27 or 28, of loose habits, had been sent to the sea-side for symptoms pointing to some abdominal disturbance. There he gradually got worse, and before long an abdominal tumour was discovered. When seen by Dr. Bristowe, he was sallow, pale, and emaciated, with an enormously distended abdomen. He vomited all food.

"There was a large, ovoid, somewhat irregular tumour occupying mainly the left side of the abdomen. It extended from the ribs above to the pubes below, and from the left loin to a couple of inches or so to the left of the umbilicus." The intestines were pushed to the right. There was no fluid in the peritoneal cavity. It seemed to be connected with the spleen, kidney, or peritoneum in the neighbourhood of these organs. It was tapped, and a gallon and a half of opaque, turbid, dark reddish-brown fluid removed. "The fluid was simply altered uncoagulated blood," showing red and white blood-corpuscles under the microscope. At the end of two months the tumour filled up again, and was retapped, fluid of the same nature being drawn off. After another two or three months he returned home perfectly well. Dr. Bristowe examined the patient several times after, and never discovered the least sign of the former tumour.

In the following case the cyst is somewhat similar in position and extent.

Case 21 (Dr. Oliver, 'Lancet,' 1884, vol. ii, p. 632).—Woman æt. 24, a widow, living the life of a prostitute. For three months suffered from metrorrhagia, after which there was cessation of menses for the eighteen months previous to admission. Three months before admission, constant pain in the left side of the abdomen led to the discovery of a swelling. "For some time, however, before the abdomen swelled she had suffered from frequent vomiting." The bowels were normal.

On admission, under Dr. Oliver, "the abdomen was greatly distended; it measured at the level of the umbilicus 40¼ inches; fluctuation well marked, particularly in front and on the left side." "The right flank and iliac region were tympanitic on percussion, but as the middle

line of the abdomen was approached the note became duller and less tympanitic. Over the whole of the lower half of the left abdomen there was absolute dulness on percussion, and there was marked resistance but no pain on pressure. The dulness extended about an inch to the right, and had its upper margin two inches above the umbilicus." On placing the patient on her right side, the left half of the abdomen still remained absolutely dull. The uterus was freely movable, nothing being felt either behind or in front. A small trochar and cannula were inserted, and 148 oz. of blood-red fluid escaped. The fluid contained red blood-cells, and a "few indefinite corpuscles like ordinary lymph-cells, some of which had undergone fatty degeneration."

The cyst filled up again, and was again tapped, 84 oz. being drawn off; but when she left the infirmary five months after admission all traces had disappeared.

The urine was at first scanty, but afterwards became more abundant. It contained a considerable amount of urates, but no albumen.

The following case is reported by Mr. Treves as a pancreatic cyst. Its claim to be placed in that category does not appear to be indisputable.

Case 22 ('Lancet,' 1890, vol. ii, p. 555).—A healthy, muscular railway-foreman, æt. 40, was admitted to the London Hospital, under Mr. Treves, July 11th, 1888. For seven or eight months he had noticed a throbbing sensation in the region of the umbilicus, which was noticed, however, only when lying on the abdomen.

He went to see a doctor for vague pains in the abdomen, who discovered "a median swelling in the vicinity of the umbilicus, about the size of two fists, rounded and free from pulsation."

At the time of admission, "in the abdomen was a tumour which, although quite median, was a little more prominent on the left than on the right side. Its upper limit was some three inches above the umbilicus, and below it reached to the pubes. It extended so far laterally as to occupy nearly the whole of the front of the abdomen." Both flanks and iliac fossæ were resonant. There was a resonant

area between the tumour and the liver. The mass felt smooth and rounded; it was firm and elastic, and exhibited no sense of fluctuation. There was no pain and no tenderness. The existence of a cyst was not suspected, the tumour being supposed to be solid. At the operation it is stated that "it was evidently behind the peritoneum," the reflections of which "from the sides of the tumour on to the kidney and mesocolon were noted." "The small intestines were for the most part below and to the left, while the omentum and transverse colon were above." After the cyst was opened, "on introducing the hand the cyst was traced up to the region of the pancreas. Its upper boundary was transverse, and it was closely attached to the spine and aorta." "This intimate association," however, extended "as low down as the fourth lumbar vertebra." On opening the cyst a large quantity of opaque fluid of brownish red colour escaped. Eighty-four ounces were collected, and it was estimated that it contained about 100. About a teacupful of brownish blood-clot was removed after the fluid had escaped. The fluid was alkaline, contained many blood-corpuscles, some albumen, but no bile nor urea.

The interior of the cyst-wall was quite smooth. The cyst was drained, and the wound healed in three months.

Excluding the cases of rapidly fatal hæmorrhage into the mesocolon and mesentery reported by Dr. Isambard Owen and Dr. Gilbart Smith, brief notes of twenty cases of cyst of the abdomen have been given in which blood has been present in the contents in varying quantities. In four cases the colour of the contained fluid has been relied upon as distinctive of blood. I do not think the absence of absolute proof necessitates their expulsion from the list.

Roughly classified as to position, five cases were situated in the upper part of the abdomen, possibly in the layers of the transverse mesocolon, three were connected with the mesentery, one in the great omentum, two apparently in the small omentum, and one in an obscure position behind the stomach. One, the last, was retro-peritoneal, and the other seven were situated at the side of the abdomen either above or below, probably partly retro-peritoneal and partly between the layers of the ascending or descending mesocolon.

*Age and etiology.*—Having glanced through these cases we may briefly summarise the leading features.

It will have been noticed that the ages of the patients vary considerably; two were in girls aged eight and eleven years respectively, while another was in a man aged seventy-six.

With so few examples statistics are of little value, but in the preceding cases they were about equally frequent in the decades from ten to forty; four occurring between eleven and twenty, six between twenty-one and thirty, and four again between thirty-one and forty, while there was only one between forty-one and fifty.

Thirteen were in males and seven in females, and two other cases in men, that will be quoted afterwards in discussing the relation of these cysts to those of the pancreas, would bring the number in the former up to fifteen.

Injury evidently frequently plays an important part in the production of sanguineous cysts, since there was a history of traumatism in six cases.

In Case 1 it preceded the appearance of the cyst by about two months, but was not at the time considered by the patient to have had any connection with it. In other cases, however, such a connection was undoubted, since pain lasted in the region struck until the tumour appeared, when there was often some abatement in its severity.

The time that elapsed between the appearance of the cyst and the injury varied considerably. In the case of Richet (No. 10) it was six months, in Michalski's (No. 17) eleven weeks, and in Dr. Sutherland's (No. 5) apparently only twenty-four hours. In the remaining fourteen cases no history is given in two, and in the others traumatism as a cause is not mentioned.

As in the cases following injury, pain is a frequent premonitory symptom. It is mentioned as preceding the appearance of the tumour in seven, and was associated with vomiting in six.

In the case of Terrier (No. 6) colic was present six months before, in that of Gallois (No. 12) seven months, and in Dr. Bristowe's case (No. 18) two months.

In several cases (Menzies, Oliver, Michalski, and that seen by myself) the distressing symptoms were relieved when the tumour appeared, but in others (Clifford Allbutt, Terrier) they continued.

The above cases show that in some cases the cyst appears to be due to traumatism, but that it follows the injury at a variable period, sometimes of several months in length, pain and perhaps vomiting being present. In others no history of injury is recorded, but, as in those following traumatism, the patient generally suffers from attacks of pain and vomiting before the tumour appears.

In three cases (1, 5, and 15) there was rise of temperature, accompanied in one by abdominal distension, thought to be general peritonitis ; in another by fulness in the epigastrium, thought to be localised inflammatory effusions.

In two cases (Nos. 2 and 11) a small tumour was noticed in the earlier attacks, which afterwards disappeared.

*Clinical situation and features of cyst.*—Perusal of the above cases will have shown that the cyst may be situated in almost any region of the abdomen. They are, however, more common in the upper than in the lower half. In five it was centrally placed, occupying parts of the epigastric, umbilical, and both hypochondriac regions, reaching down to or just below the umbilicus. In two (Nos. 1 and 2) it was rather more prominent to the left of the median line.

Other cases are described, occupying mainly either the right (Nos. 13 and 14) or left (No. 17) half of the upper part of the abdomen, nearly the whole of one side (Case 18), or the lower half (Case 19). Again, the cyst may be centrally placed in the lower half of the abdomen ; *e. g.* it is stated that had one tumour (Case 6) been present in a woman instead of a man it would have been considered an ovarian tumour. The cyst in Case 7 was similarly placed, but there was resonance below as well as above. Another case (No. 9) was mistaken for a distended bladder. Wherever situated the tumour is smooth, generally tense and elastic, but sometimes described as fluctuating. Its mobility depends largely upon its position, those centrally placed being mobile while those at the sides of the abdomen appear to have been fixed. When in the upper part

of the abdomen, pulsation may be communicated from the aorta.

*Clinical course.*—Having reached a certain size the tumour generally becomes stationary, and although vomiting and abdominal pain may have ceased it still appears to injuriously affect the patient's health, since wasting, associated with a sallow complexion, has been common.

On cure of the cyst, however, the return to health has been rapid, weight being quickly regained.

*Terminations.*—Natural cure by opening into the intestine occurred in two cases; in one (Case 2) after three aspirations, but in the other (Case 3) three or four days after a few ounces only had been withdrawn. Another case (No. 12), ended fatally from peritonitis, following rupture into the peritoneal cavity.

In Case 1 there was vomiting of a considerable quantity of blood, said by a very reliable nurse to have been as much as four pints; and yet, curiously enough, there was no diminution in the size of the tumour. The amount was probably over-estimated, but it must have been large. Vomiting of blood in much smaller quantities occurred afterwards, and a clot was passed by the rectum.

Left without surgical interference, one cyst existed for twelve years, the man, aged forty-four, dying of cholera (No. 9). In another (to be afterwards mentioned) the tumour was present for six years, the patient dying eventually of tuberculosis of the lungs, with thrombosis in the superior longitudinal sinus.

*The cyst fluid.*—The fluid withdrawn from the cysts is most commonly described as reddish-brown, but is also said to have been chocolate, like light café noir, and once or twice red, or blood-red in colour.

In all cases microscopically examined there has been a deposit of red blood-cells, often associated with granules of colouring matter, and both frequently in considerable quantity. Leucocytes have been commonly present, and in two instances larger cells, described in Dr. Clifford Allbutt's case as of epithelial type. Phosphates are mentioned in one case.

*Morbid anatomy.*—We have abstracts of six cases that

terminated fatally. Of these, one was in the mesentery, one in the great omentum, one in the small omentum, two apparently partly between the layers of the small omentum and partly behind the stomach, and one partly retro-peritoneal and partly between the layers of the mesocolon near the hepatic flexure.

The cyst-wall is described in one case as fibrous and rather thin, in another as somewhat thick and resistant. The internal surface may be furrowed, flocculent, and covered with ecchymoses (10) ; or covered with layers of reddish-brown, granular, coagulated blood (9, 14).

In Case 9 there was in addition a greyish friable mass, described as like brain substance macerated in alcohol, furrowed by channels containing blood. In two cases recent hæmorrhage had occurred, in one (12) the cyst being filled with dark blood clot; in the other (14) a black clot lay beneath the older reddish-brown granular layers, and other clots were in the centre of the cyst.

The surrounding blood-vessels, when carefully examined, were said to present nothing abnormal (Cases 9 and 14). The surrounding organs, when mentioned, are said in the majority of instances to have been healthy. In Case 10 the pancreas, spleen, and kidneys are all said to have been healthy. In Case 12 again, although the cyst was situated behind the stomach, it is distinctly stated that it was separate from the pancreas, spleen, and transverse colon, and that it stopped a little distance from the liver. In Case 14 the liver was pushed to the right, the cyst coming in contact with the diaphragm. Although adherent to the liver, it was quite distinct from it. The liver in this case is described as pale and fatty.

Of organs at a distance, the heart is described as pale and flabby in one case (No. 14), healthy in another (12), but in this case both upper lobes contained tubercles, with small cavities at the apices. The kidneys also showed parenchymatous nephritis.

In Case 11 the pylorus was pressed upon, and the stomach hypertrophied. Near the pylorus, underneath the mucous membrane, there was a small cavity the size of a hazel nut containing blood, which communicated by a tortuous channel

with the cyst behind. This is interesting, taken in connection with the history of those cases which terminated in natural cure by escape into the intestines. Possibly this was commencing to open into the stomach.

Microscopically examined, the cyst-wall is stated to have been composed of connective or fibrous tissue with flat cells in one case (No. 10), fibrous tissue with embryonic cells in another (No. 11). In Case 12 the recent hæmorrhage was thought to have come from the cyst-wall in the neighbourhood of the stomach; and in Case 14 vascular channels are described, freely communicating with one another.

The two cases of death from hæmorrhage into the layers of peritoneum brought before the Medical Society by Dr. Isambard Owen and Dr. Gilbart Smith (Cases 2 and 9) may also be briefly considered, since they appear to be closely allied to the above. In both the source of the hæmorrhage was carefully sought for, but could not be found; the blood-vessels and abdominal organs seem to have been quite healthy. In Dr. Owen's case the heart was flabby and the mitral valves were thickened. There were also some tuberculous nodules in the lungs. There was nothing, however, that threw any light upon the case.

*Pathology.*—This is obscure. In a considerable percentage of cases, as we have seen, the cyst follows at a variable period after an injury, but how the injury acts is not clear.

It is, of course, possible that small hæmorrhages between the layers of the peritoneum may follow directly upon a blow, but it is more common for such hæmorrhages to lie between the peritoneum and the abdominal wall. In rare instances a hæmorrhagic peritonitis is set up, and small blood effusions, limited by adhesions, may be found in the peritoneal cavity. In the report of one such case,[1] in addition to small collections of blood between the intestines, one of which reached the size of two fists, there were small hæmorrhages in the layers of the great omentum. The blood effusions both within and without the peritoneal cavity were probably secondary to inflammation. Whether extensive hæmorrhages do or do not follow directly upon a blow has, however, no very marked bearing upon the question before us, since the cysts we are considering, when due

[1] Lannengue, 'Bull. de la Société anat.,' 1865.

to traumatism, as a rule follow at an interval varying from a few days to several months.

Rouiller suggests that a "pachy-peritonitis" is set up by hæmorrhage occurring from the new vessels of the false membranes. If this view were correct we should expect to find several cysts rather than one solitary one, or at least there would be thickening of the peritoneum around the cyst. Another still more important point is that the cyst would be inside the peritoneal cavity, and not between the layers of the peritoneum as in all probability it was invariably situated when not retro-peritoneal. In two cases the reflections of the peritoneum from the surface of the cyst were carefully noted, and in others there seems to have been no doubt that the cyst was situated between its layers.

It is possible that a lesion of an artery might take place, leading to the formation of an aneurism which afterwards ruptures. It is improbable, however, that a vessel could sustain such an injury and yet the intestines completely escape, but allowing such a possibility, the aneurism would be almost as likely to rupture into the peritoneal cavity as between its layers, if situated (as we must suppose it would be in the majority of instances) between those layers.

Thrombosis of either a mesenteric vein or artery is a possible cause, and in connection with the possibility of such an origin the following case may be of interest.

M. Pilliet, 'Bull. de la Société anat.,' 1889.

A strong, athletic-looking, but very stout man, æt. 72, was seized with sudden abdominal pain while sawing wood.

Vomiting and swelling of the abdomen soon followed. The symptoms resembled those of acute intestinal obstruction, and he died two days later.

At the post-mortem a portion of the small intestine about 40 cm. long was found swollen with blood effused into its walls, while hæmorhage into the portion of the mesentery connected with it formed a hard cake 2 cm. in thickness. The mesenteric veins of that part of the mesentery contained ante-mortem clot, and microscopically all the coats of the intestine except the internal were unrecognisable owing to the infiltration with blood-cells. The epithelium had disappeared.

Although in this case venous thrombosis had given rise to hæmorrhage between the layers of the mesentery, the small intestine was also seriously affected, and this, of course, is only what we should expect. The reports of four other cases that I have read show this effusion of blood into the coats of the bowel to be a marked feature, while hæmorrhage between the layers of the mesentery is either absent or comparatively slight and unimportant. Again, experiments on animals show that blockage of a mesenteric artery by thrombosis or embolism is not likely to be a cause, since occlusion of one of these vessels causes still severer lesions of the coats of the bowel, the mesentery being comparatively unaffected.

We may, therefore, dismiss vascular embolus or thrombosis from further consideration.

Rouiller suggests a separate pathology for those cysts in which there is no history of traumatism. In this class he supposes that the hemorrhages are secondary to a sclerosis of the peritoneum due to alcoholism, and adduces in support of his view two cases of peritonitis associated with cirrhosis of the liver, in which there were numerous small encysted collections of blood.

Strongly militating against this view is the fact that several of the cases without history of injury have occurred in comparatively early life, one at the age of eleven years, two at twenty, and one at twenty-two. The girl of eleven, now six years after, is a healthy young lady, and the boy of twenty, eight years after was in perfect health. It is true that in rare cases cirrhosis may be present at the above ages, but one would hardly expect the subjects of the disease to be in good health several years after.

It must also be mentioned that in none of the post mortems has cirrhosis of the liver been mentioned.

I have criticised adversely the views of M. Rouiller, and have given some reasons for doubting whether the cysts can be due directly to injury or indirectly through aneurism or thrombosis of a blood-vessel. We will now proceed to see whether it is possible that the nervous system can be concerned in their production.

The frequency with which dyspeptic disturbance, often

taking the form of pain, constipation, and vomiting, precedes the appearance of the tumour has already been pointed out. These prodromal symptoms may occur with or without a history of traumatism. When a history of injury is present the symptoms generally become well marked immediately, and although after a few days their severity lessens, they remain present in greater or less degree up to the time of the appearance of the tumour. It is obvious that some disturbance has been set up that finally culminates in a blood effusion. Of the nature of this disturbance we can only surmise, but, as remarked above, we will consider whether the nervous system can be concerned in the matter.

Perhaps the possibility of the nervous system playing some part in certain hæmorrhages is not frequently considered. In hæmatemesis due to cirrhosis of the liver, for example, why should the blood rush forth with such remarkable and often fatal suddenness? If due to congestion pure and simple there should be gradual but continued oozing. Again, may not the nervous system be concerned in the epistaxis of childhood and adolescence (the blood-vessels can hardly be at fault)? and may not hæmophilia be an inherited weakness of the nervous mechanism of the vascular system? When suggesting a nervous cause for sanguineous cysts to Dr. Goodhart, he very kindly mentioned the occurrence of hæmorrhage into the supra-renal corpuscles as bearing upon this view. Apparently hæmorrhage into these organs is not so very uncommon, and the curious point about the lesion is that it occurs on both sides at the same time.

A brief reference to a case may be of interest.

Dr. Pritchard, 'Lancet,' 1890, vol. i, p. 750.—A demented old woman, æt. 76, with irregular heart, was admitted into the County Asylum, Lancaster. She complained of aches and pains, but was up and about.

February 15th, 1890.—She was suddenly seized with severe abdominal pain. She vomited several times and became collapsed. She did not pass any urine for twenty-four hours, and then only a small quantity, which was normal. The vomiting continued, and she complained of great pain in the abdomen, first in one hypochondrium, then in the other, and sometimes in the lumbar region. The vomiting

stopped on the third day, but the bowels were not opened. On February 5th she developed pneumonia, and died on the 6th.

*Post-mortem.*—An extravasation of blood, about the size of two kidneys, surrounded the kidney on the right side. This was found to have escaped from the right supra-renal capsule, which was swollen to about two thirds the size of the kidney. The hæmorrhage, however, was not limited to that side, for it is stated "that the same condition of things existed on the left side, the supra-renal capsule being swollen to the same extent."

Here we have extensive symmetrical hæmorrhages; if an isolated case, we might think it a mere coincidence, but when hæmorrhage in this situation is found at all it is almost invariably two-sided. Such hæmorrhage can scarcely be due to double local independent lesion of the blood-vessels. There must be some cause acting either through the blood or the nervous system. It is possible that there may be a susceptibility of the two capsules to some poison in the blood which produces an acute hæmorrhagic inflammation, but against such a view is the fact that microscopically the structure of the supra-renal capsules which surrounds the clot has been found normal. This being the case, vague though the idea may appear, we are almost driven to suppose that the hæmorrhage is dependent upon some affection of the nervous system.

Dr. Rolleston, when reporting two cases of fat necrosis of the peritoneum at the Pathological Society on October 18th, 1892, suggested that the degeneration was due to some lesion of the solar plexus spreading from a diseased pancreas. In one case there was an abscess in the head of the gland; in the other, however, there was no evidence of pancreatitis, although there was hæmorrhage around the pancreas in addition to the fat necrosis. In this case, as in some other recorded cases of fatal lesions of that gland, there was a history of injury, followed by attacks of abdominal pain. At the onset of the fatal illness the patient suffered from pain, vomiting, and constipation. Dr. Rolleston laid stress on these symptoms as lending support to his view of some affection of the solar plexus as a cause for the fat necrosis of the peri-

toneum. Assuming this part of his view to be correct, in the second case the nerve lesion must have been primary, and not secondary to disease of the pancreas, since that gland was apparently healthy.

A brief abstract of the case may be of interest.

A man æt. 30. One year and a half previously a cart had passed over his abdomen. Several attacks of abdominal pain since. After dinner had sudden abdominal pain, vomiting, and constipation, with collapse. Laparotomy. Death thirty-six hours afterwards.

*Post-mortem.*—Hæmorrhage around the pancreas, the source of which was not manifest. Disseminated fat necrosis in the abdomen. There was no evidence of pancreatitis.

There is a striking similarity in the history to some cases of sanguineous and also of pancreatic cyst. An injury, followed by abdominal pain, finally ending in sudden hæmorrhage. It is not to this, however, that we wish to draw attention, but to the fact that it was suggested that the pathological condition discovered might be due to a lesion of the solar plexus.

Having glanced at the above case, the following is of great interest. It is quoted from Frerichs by Fitz in the 'Boston Medical and Surgical Journal,' 1890, vol. cxxii, p. 573:

A man æt. 41, a brandy drinker, who suffered from dyspepsia, had attacks of pain in the epigastric region and died in sixty hours.

At the post-mortem the pancreas was large and flabby; there was a small abscess in the head of the pancreas, and fatty streaks elsewhere. Below the liver was a large reddish-brown tumour, extending from the gall-bladder to the spleen. It consisted partly of fluid, partly of clotted blood, effused between the layers of the small omentum, the hepato-duodenal and gastro-splenic ligaments, and the transverse mesocolon. It extended down to the kidneys and along the pancreas, from the portal vein to the spleen. The source of the hæmorrhage could not be found.

I should be inclined to suggest that both the pancreatitis and the hæmorrhage around the gland were due to a lesion elsewhere, but it is to the blood effusion only that I wish to refer.

Here we have an extensive hæmorrhage, situated mainly between the layers of the peritoneum. It hardly seems credible that a hæmorrhage in the immediate vicinity of the pancreas can have mounted upwards, separated the peritoneum from a portion of the under surface of the liver, and have invaded the small omentum. Since no source could be found for the hæmorrhage, it is possible that the blood may have been effused not from one vessel, but from many, and those vessels situated where the effusion was found.

The probability of this being the case is shown by two of the cases shown at the Medical Society in 1881, and already quoted. In Dr. Isambard Owen's case (No. 2) the transverse mesocolon was distended by an enormous blood effusion, but nothing was discovered to explain its occurrence. All the blood-vessels appeared healthy, as well as all the abdominal organs, and presumably the pancreas amongst them. In Dr. Gilbart Smith's case (No. 9) the extravasation was into the mesentery, but nothing abnormal was discovered.

If under certain circumstances small areas of fat necrosis become dotted over the peritoneum, it is possible that under somewhat similar circumstances changes may take place in the smaller blood-vessels that will allow the escape of large quantities of blood. If trophic changes can take place in the peritoneum itself, why should they not in the blood-vessels? Such a change occurring rapidly in the smallest blood-vessels and capillaries over a wide area would readily give rise to blood effusion.

At the commencement some stress was laid upon the history of traumatism, and some inquiry as to the connection between the injury and nerve-lesion may suggest itself.

We are apt to forget what a profound effect a peripheral impression may have upon the nervous system. The sudden arrest of an epileptic fit at its onset, by firm pressure upon the site of the aura, is an example of the influence that can be brought to bear upon the central nervous system. Still more striking is the fact stated by Dr. Gowers, that progressive muscular atrophy and paralysis agitans may both follow an external injury; in one case the wasting, in the other the tremor, commencing in the part injured. An

illustration of this interesting fact has been shown by a case recently under Dr. Hale White in John Ward, Guy's Hospital. A man with paralysis agitans had been stabbed in the palm of the hand, the tremor beginning there soon after.

It is possible that the lesion we have supposed to exist may be started in a similar way. It manifests its presence by abdominal pain, vomiting, and constipation, and finally leads to sudden blood effusion.

When there is no history of injury, the nerve-lesion must of course have some other origin.

It must be mentioned that somewhat militating against the view that a nerve-lesion is the cause of the hæmorrhage, is the fact that when the cyst has been cured the symptoms as a rule disappear, and the patient enjoys perfect health.

The only explanation that I can offer is the following:—The nervous lesion, although we must suppose it to be comparatively central, is small, and only influences the sympathetic over a limited area—the area into which hæmorrhage takes place. The hæmorrhage having taken place, the nerves of that area are destroyed by the tension of the cyst. These nerves, or their terminations, having been destroyed, the lesion although still existing fails to make its presence felt.

It may be argued that the early symptoms are due to blood effusions, not sufficiently large to attract attention. This view would be supported by the fact that in a few cases these early symptoms were accompanied by a small tumour that afterwards disappeared. Adopting this view, the abatement of symptoms when the cyst fully develops would have to be explained by irritation of the sympathetic nerves by a small effusion, and destruction by a large. Allowing that such a cause for the symptoms cannot be excluded, the cause of the hæmorrhage would still remain unexplained.

*Treatment.*—Of those treated surgically, aspiration or tapping was used alone in eight cases. In four of these a single operation was sufficient, in two the operation was repeated once successfully, in one the cyst partially refilled after a second aspiration, but was permanently cured by bursting into the bowel, and in the eighth, in a man æt. 76, death took place, apparently from pleurisy, but not obviously as the result of treatment. In four cases injection of some

fluid of caustic or antiseptic nature was associated with tapping. Of these two died, in one the fluid entering the peritoneal cavity instead of the cyst. Laparotomy, with stitching of the cyst to the abdominal wall and drainage, was successful in two cases. In four the operation of extirpation was decided upon, two of which recovered; the others ended fatally.

*The relation to cysts of the pancreas.*—Should the reader have carefully read the reported cases of pancreatic cyst, he cannot fail to have noticed the resemblance between some of them and the earlier cases quoted in this paper. The shape and situation of the cyst is similar, they both contain blood, they both are preceded by a history of dyspeptic disturbance, in both there may have been a history of injury.

It may be suggested that the cases in the upper part of the abdomen recorded as sanguineous cysts were really connected with the pancreas, but that the reactions of pancreatic juice were not looked for, and consequently not discovered. Although in several cases recorded as pancreatic cysts those reactions have not been present, it must be admitted that it cannot be definitely stated that they were absent in these cases of sanguineous cyst. It has, however, been pointed out that sanguineous effusions may be present in any region of the abdomen, and it would be unreasonable to class some with the pancreas merely because they happened to occur in the vicinity of that gland. Instead of jumping to that conclusion, we may conceive it to be possible that the pancreas may be secondarily affected by a blood effusion in its neighbourhood. The presence of blood in a pancreatic cyst is commonly explained by hæmorrhage into a cyst already present, which perhaps owes its existence to blockage of the duct, but I propose to take the opposite view, that an effusion primarily sanguineous may soon fail to give marked evidence of the presence of blood, and that the pancreatic cyst originates in a blood effusion not necessarily arising in the gland itself.

Before proceeding further, it may be as well to quote a few brief abstracts of cases recorded as pancreatic cyst, in order that it may be clear that blood is frequently present.

In the following cases, while noticing the characters of the fluid, the situation of the tumour and the history are also worthy of attention.

The fluid either obviously contains blood, is brown in colour, or is stated to deposit red blood-cells. As noted in two of the cases of sanguineous cyst, large cells may also be present.

As to the history, the tumour is sometimes preceded by a period of abdominal pain; in others it appears almost without warning.

The position of the cyst will be noticed to resemble that of the cases of sanguineous cyst in the upper part of the abdomen.

The first two cases are of comparatively sudden onset (Gussenbauer, quoted by Senn, 'American Journal Med. Sciences,' 1885, vol. xc, p. 25). A man, æt. 40, was taken suddenly ill after having indulged too freely in the pleasures of the table. He had nausea, pain in the stomach, and vomited repeatedly. In a few days the symptoms passed off, but he discovered a swelling in the region of the stomach, which increased rapidly in size, and after two weeks caused bulging in the epigastric region. During the next month he vomited, and become emaciated. When seen ten weeks after the onset there was a tumour "which extended over the right and left supra-umbilical regions, and from the left costal arch into the right hypochondriac region." On the middle line the tumour measured $7\frac{7}{8}$ inches from the xiphoid cartilage to two inches below the umbilicus. The cyst was opened and drained, the fluid, of greyish-black colour, having in suspension large quantities of pigment and a few red blood-corpuscles in a state of degeneration. It contained no bile-pigment, metalbumen, peptones, or sugar. The skin became macerated by the fluid that escaped from the wound, which digested albumen, and turned starch into sugar.

A very similar case is that of Thiersch, also quoted by Dr. Senn (ibid., p. 29).

A man æt. 38, of previous good health, was taken ill suddenly while at work. He complained of nausea and lassitude, and a tumour developed in the region of the stomach. The rapidity with which the tumour appeared and the dis-

tinct fluctuation led the attending physician to diagnose an abscess in the anterior abdominal wall. An incision was made in order to evacuate what was thought to be pus, and a cystic tumour was discovered. Two and a half weeks after incision was made, and three quarts of a chocolate-like fluid escaped. A fistula remained, which discharged thin serous fluid. Examination for pancreatic fluid, however, gave negative results.

The above case does not appear to possess any substantial claim to be called a cyst of the pancreas. Its resemblance, however, to the case that precedes it, in which fluid having the properties of pancreatic juice was discharged from the wound, is certainly striking.

The following brief abstracts are taken from Dr. Newton Pitt's paper on a case of pancreatic cyst, read before the Royal Medical and Chirurgical Society, June 9th, 1891.

*Lardy.*—A man, æt. 37, a year previously was seized with a sudden attack of abdominal pain, which lasted for some hours. After a second attack he noticed a fulness in the epigastrium. He wasted greatly. A cyst was present, in front of which were the stomach and colon. It was punctured, and a dark-red bloody fluid was drawn off. It contained red blood-cells, large fatty cells, and cholesterin.

*Subotic.*—Man, æt. 20, was seized with attacks of colic and vomiting. An abdominal tumour had been noticed two years previous to his admission, and for one year he had frequent attacks of vomiting. On admission the patient was anæmic and emaciated. There was a tumour the size of a child's head, which extended two fingers' breadth below the umbilicus. The cyst was drained. A turbid, brownish, alkaline fluid was evacuated, containing fatty nucleated cells and red blood-cells.

*Wölffler.*—A girl æt. 21, with acute gastritis, a history of jaundice two years previously, followed by abdominal tumour. There was no history of injury. The tumour occupied both hypochondria. There was tympanitic resonance above due to the stomach, below due to the colon. The cyst was opened and drained. The fluid was brown, turbid, contained red and white blood-cells and elliptical

epithelial cells, with fat drops and cholesterin crystals. It also contained a trace of peptone and a sugar ferment.

*Steele.*—Man æt. 40. At the age of twenty-seven he was crushed between two railway waggons. In the following year a cyst gradually developed in the umbilical region. This was aspirated, and fifty-two ounces of chocolate-coloured fluid were drawn off. This aspiration was repeated four times during the succeeding ten years.

*Riegner.*—Girl, æt. 23, fell from a great height, after which she suffered from attacks of abdominal pain. Three years later the pain became more severe, she became slightly jaundiced, and had severe attacks of vomiting. An epigastric swelling which steadily increased in size was observed. This was formed by a dilated stomach over a cyst. The removed fluid was brown, containing red and white blood-corpuscles; it emulsified fats, and was amylolytic. The fluid that escaped from the fistula after the operation digested fibrin.

In the two last cases there was a history of injury, and in connection with this fact we may remind the reader of the two cases of Mr. Jordan Lloyd quoted early in this paper. In both an epigastric tumour followed an injury, and in both the fluid contained blood. In the second case it also possessed amylolytic action and emulsified fats.

In the above cases it will have been noticed that either blood was present or that the colour very strongly suggested it.

In four the properties of the fluid gave evidence of connection with the pancreas, in three those characters are not mentioned, and in two, although searched for, could not be found. It is not desirable to multiply cases, but the following, quite recently reported,[1] is interesting in this connection: The patient was under the care of Mr. Paul Swain, of Plymouth. A tumour present between the umbilicus and the xiphoid cartilage was opened and drained by Mr. Swain. The fluid was of dirty brown colour, contained red blood-cells, and a few (?) colostrum cells, but Mr. W. L. Woollcombe, to whom the report is due, very kindly answered a letter of mine, saying that it possessed none of the properties

[1] 'British Medical Journal,' March 4, 1893.

of pancreatic juice. The irregularity of the presence of these properties in the recorded cases of pancreatic cyst is worthy of note.

It has been pointed out that sanguineous and pancreatic cysts contain fluid that is often almost identical; in both there may be little else but blood or a brown fluid containing red blood-cells, and in both larger nucleated cells may be present. The import of these nucleated cells is, perhaps, not altogether clear. Similar cells are frequently present in the deposit of pleural and peritoneal effusions, and are presumably derived from proliferation of the endothelial layer of the serous membranes. The following suggestion is brought forward with some diffidence, but it seems possible that the presence of these cells in the fluid of cysts known as pancreatic, may point to a connection with the peritoneum rather than with the pancreas, irritation of effusion between its layers producing similar proliferation to that which takes place on the surface in chronic peritonitis. In favour of this view is the fact that microscopic examination of the wall of one of the sanguineous cysts showed flat cells mixed up with the fibrous tissue.

In situation and shape sanguineous cysts of the upper part of the abdomen also closely resemble pancreatic cysts. In both pancreatic and sanguineous cysts the lower limit is frequently situated an inch or two above or below the umbilicus.

Early in this paper reasons have been given for believing that both sanguineous and pancreatic cysts may be situated in the transverse mesocolon, and the remarkable case of death from hæmorrhage in that position, reported by Dr. Isambard Owen, on the one hand, and the cyst of Salzer on the other, would strengthen this conclusion.

Although other fluids besides digestive juices will emulsify fats and turn starch into sugar, the irritation of the wound from the fistula left after the operation in some cases of pancreatic cyst, and the fact that this fluid digests fibrin in others, points to a definite connection with the pancreas.

The exact relation, however, is still obscure. That the cyst generally starts as a retention cyst is highly improbable. Mr. Targett first directed my attention to the possible connection

of sanguineous cysts with those of the pancreas, remarking that if the latter originate by blockage of the ducts, smaller cysts of various sizes ought to be more common in patients dying from various diseases. Dr. Goodhart has also remarked that the same question has arisen in his mind.

I have suggested that a pancreatic cyst may originate as a blood effusion in the neighbourhood of the gland, not necessarily arising in the gland substance, it being supposed that the tension of the sanguineous effusion may cause atrophy of part of the gland substance, and escape of fluid from opened ducts.

In the following case a sanguineous cyst is adherent to a partially atrophied pancreas. It must be admitted that it cannot be denied that the cyst may have originated in the pancreas itself; but a case we shall quote afterwards, in which a blood effusion destroyed the left kidney by pressure, will show that it is possible or even probable that the gland structure was destroyed in a similar way.

Merigot de Treigny ('Bulletins de la Société anatomique,' Paris, 1885).—A jockey, æt. 32, had noticed six months previously, during a dyspeptic attack, the existence of a hard tumour limited to the epigastrium. He could not remember any definite blow in that region, but in following his calling he had frequent falls. For a fortnight the tumour remained of considerable size, and was associated with pain and nausea. It then diminished rapidly, and the patient could only feel a mass the size of a chestnut. From time to time he had shooting pain, which was relieved by pressure on the epigastrium. Recently, however, he had suffered no pain, but the tumour increased and rendered riding impossible.

On admission, a smooth rounded tumour was situated at the junction of the left hypochondrium with the epigastric region. It was separated from the xiphoid cartilage by a small interval, and descended to about two fingers' breadth from the umbilicus. It did not appear to encroach upon the costal margin, and was moveable from side to side. It was elastic, with a sense of fluctuation. Above and to the right was stomach resonance. Exploratory puncture drew off a highly albuminous, brownish fluid, showing altered and deformed red blood-cells and leucocytes under the micro-

scope. The cyst was incised and drained, but was followed by death.

*Post-mortem.*—A cyst about the size of two fists lay between the stomach and transverse colon, the latter encircling the tumour in front and below. The third part of the duodenum was below and the pancreas behind. It is stated "the pancreas was distinctly seen in its right half; its head showing nothing noteworthy, but on approaching the tumour, it was seen to be closely united with the fibrous tissue, its lobules merely compressed at certain points, but further on disappearing in the sclerous matrix, and immediately behind the tumour no trace could be discovered. In the left part some lobules were to be recognised, but the tail was deformed, atrophied, and lost in the midst of the fibrous tissues."

The splenic artery and vein, however, were unaffected. "The posterior surface of the cyst was crossed by the splenic artery, which was absolutely healthy, adhering only by cellular tissue, and did not present any trace of old rupture." Lower down the vein was seen closely applied to the posterior wall, and more intimately adherent.

Here we have a sanguineous cyst adherent to the pancreas, which is atrophied at the position of contact.

It is of course quite conceivable that the cyst originated as a hæmorrhage in the gland itself, and Dr. Senn has proposed such an origin for these cysts. He suggests:—1stly. Hæmorrhage into a pre-existing pancreatic cyst; 2ndly. Parenchymatous hæmorrhage producing a cyst, followed by hæmorrhage from the cyst-wall; 3rdly. That the hæmorrhagic cyst may originate in a dilatation of one of the vessels of the pancreas.

A reason has been given for thinking the first somewhat improbable, but we cannot exclude the other two. Two cases, however, in which there was some evidence that the kidney became attached to a blood cyst and was secondarily affected by pressure, suggest that a connection with the pancreas may be established in a similar way. The second case, that of Mr. Marshall, is not sufficiently conclusive to warrant much stress being laid upon it, but the first is of value, since we have the report of a post-mortem examination.

J. Stein ('Archiv für pathologische Anatomie,' from the thesis by Rouiller).—A man æt. 44, who had had an apoplectic attack when fourteen years old, and had suffered from gangrene of the middle and index fingers of the right hand. In 1863 he had an attack of peritonitis. During convalescence a tumour was discovered, the size of an adult head, in the umbilical and hypogastric regions. It was immobile, smooth, and elastic. It was thought to be a lipoma. Six years after, he died with signs of pleural effusion at the left base. The tumour was unaltered.

At the autopsy the greater part of the abdomen was said to be occupied by the tumour, situated mainly to the left of the median line, but encroaching upon the right side. It was covered by the great omentum, and adherent to all the neighbouring viscera, including the aorta. Withdrawn from the abdomen the tumour was spherical, and the size of an adult head. When opened, 2 or 3 litres of a serosanguineous black fluid escaped, which on standing separated into two layers, a lower dark one and an upper of serum almost as clear as water. The lower was composed of deformed red blood-cells and leucocytes in a state of fatty degeneration. The wall of the cyst was 5 mm. thick, and composed of three layers, an outer, thin, shining, and polished, of the nature of a serous membrane; a middle, as white as porcelain, and of the consistence of fibro-cartilage, while the internal cyst was composed of an irregular layer of connective tissue. There were no blood-vessels in the wall. The left kidney was adherent to the tumour, compressed, and sclerosed; the right kidney hypertrophied, but of normal structure. The spleen was also adherent, and between it and the cyst was a small collection of pus the size of a nut.

The lungs were tuberculous with thickened pleuræ; the heart flabby, the aorta atheromatous. In the superior longitudinal sinus was a thrombus.

The presence of the small collection of pus is difficult to explain, but has no bearing upon the case. The interesting feature is the unilateral atrophy of the kidney, evidently due to pressure. The pancreas can hardly be more resisting than the kidney, and when a blood effusion is found to be connected with it secondary atrophy cannot, therefore, be

excluded. During such atrophy some ducts might be opened into the cyst, allowing escape of pancreatic fluid, and the discovery of the reactions of this fluid in a sanguineous effusion lead to the supposition that the cyst arose in the gland.

I do not wish to attach much importance to the following case, but it is worthy of mention. Here we find fluid, supposed to be urine, escaping from a drainage-tube inserted into the cyst.

Mr. Marshall ('Medico-Chirurgical Transactions,' 1883, vol. i, p. 311).—A girl, æt. 13, was knocked down by a cart, the wheel passing over her abdomen. She suffered from pain in the left loin for nine months. At the end of that time she was admitted into University College Hospital, where a fluctuating tumour occupying the left half of the abdomen was discovered. Under ether the cyst was tapped with a large trochar and cannula and a small drainage-tube inserted. Thirty-six ounces of chocolate-coloured fluid came away, which was found to contain ·33 per cent. of urea. During one of the dressings two ounces of clear fluid, thought to be urine, escaped. Four months after she was discharged cured.

The presence of urea in the fluid does not necessarily point to a connection with the kidney. Could, however, the observation as to the escape of urine be relied upon as unquestionable, it might be taken to support the evidence afforded by the preceding case.

In the history of injury followed by pain and the appearance of a cyst at a long interval, the case resembles other examples of sanguineous abdominal cyst. The connection with the kidney or ureter was, if present, probably secondary. Allowing this view to be correct, we can understand how the discovery of pancreatic fluid escaping from the wound left after the operation for the cure of blood cysts may have led to the belief that the cysts arose in the pancreas.

It is possible that, although originating as a blood effusion, the contents may become less and less distinctly sanguineous, until the fluid fails to give any indication of its origin.

The following case has some bearing upon this point.

An epigastric tumour followed shortly upon an injury, and was found to contain blood at the first aspiration. At the second dark sage-green fluid was drawn off.

Mr. Littlewood, of Leeds ('Lancet,' 1892, vol. i, p. 871).—The patient, a man æt. 30, was thrown from his horse, the horse stamping upon his abdomen with his hind feet. Thirteen days after a swelling was noticed in the left upper umbilical and lower epigastric regions.

The swelling was explored and a syringeful of dark blood removed. The swelling increased in size, at first slowly, and then more rapidly. The patient suffered a good deal of pain in that region. Seven days later, August 12th, the swelling was again aspirated and ten ounces of fluid removed, alkaline, dark sage-green in colour, sp. gr. 1015. In the afternoon the abdomen was opened in the middle line. The cyst was at once seen, having displaced the stomach upwards and the colon downwards. It was covered by the omentum. The cyst was opened and drained. A sinus formed which closed at the end of two months.

Analysis of fluid collected ten days after the operation was made by Dr. Clarkson and shown to contain serum-albumen, salts, trypsin, amylopsin, steapsin, and a milk-curdling ferment metacasein.

The alteration from blood to fluid of a sage-green colour suggests that pancreatic cysts in which little or no blood has been found may have commenced as a sanguineous effusion. It is not desirable, however, to lay too much stress upon one case, yet it cannot be denied that what evidence it affords seems to point in that direction.

Whether the hæmorrhage really commences in the pancreas itself or in its neighbourhood is not of very great importance. That it may commence in the gland can hardly be disputed, since in the cases of Störck and Parsons (quoted by Senn) sanguineous cysts were connected with the pancreas, the gland tissue in both cases being dilated by hæmorrhage that had taken place in its interior.

This fact may appear to nullify the preceding suggestion. It must be admitted that the subject is intricate, but we should be inclined to suppose that the cyst may arise from blood effusion either within or without the gland. It is at

least certain that hæmorrhage may occur into the substance of the pancreas, as in the cases collected by Fitz; in the neighbourhood of the pancreas, as in one of Dr. Rolleston's cases and in that of Frerichs; and at some distance from the gland, as in several cases quoted in this paper.

No great stress can be laid upon indefinite symptoms, but it is worthy of note that in all the three varieties above mentioned the objective part of the disease may be preceded by complaint of dyspeptic trouble. In all three the causation of the hæmorrhage is obscure. It is possible that all may have the same pathology, and that both pancreatic hæmorrhage and sanguineous cysts may be secondary to a lesion of the sympathetic system, the site of which I cannot presume to suggest.

# GLYCOSURIA AND SURGERY.

By G. BELLINGHAM SMITH & HERBERT E. DURHAM.

[Communicated by the Editors.]

## Part I.

### *Introductory.*

The object of this paper is (1) to define as far as possible the limits of operative measures in the subjects of glycosuria; (2) to show what results glycosuria exerts on the course of disease and traumatism; and (3) we shall incidentally have to point out some of the relationships existing between surgical diseases and injuries and glycosuria.

*Diabetes and glycosuria.*—The great difficulty we have to deal with is the classification of cases. Various attempts have been made to distinguish between diabetes mellitus and glycosuria; but such attempts fail, for the reason that diabetes mellitus is not a unique process. What we know about the malady shows that not only does it vary enormously in its evolution, but that its pathology probably includes several distinct lesions; and although glycosuria may be divided into transitory, intermittent, and persistent (these two latter being considered commonly as diabetes), our cases will show how hard it is to draw a line anywhere. Nor in many instances are we able to say in a given case to which class it will ultimately belong.

Such difficulties have led some surgeons to reject any attempt at classification.

Verneuil, addressing the Société de Chirurgie of Paris in 1884, said, "I hardly admit the separation [into glycosuria and diabetes], no one having, so far, shown me clearly where glycosuria (a benign condition) ends, and where diabetes (a severe condition) begins. To-day, in the possession of more facts, I have decided to discard absolutely, as a surgeon at all events, the existence of an arbitrary division which does not present, in my opinion, any practical utility. . . . From the surgical point of view the presence of glucose in the urine, whatever its amount, is always a serious fact." And Tuffier, in 1888, supports this opinion. He attempts no classification; he believes "that the simple presence of glucose in the urine has been sufficient to provoke grave complications."

*Classification of cases.*—Although we readily admit the above difficulties, we think it of importance that some distinction should be made. In order to place the question upon a reasonable and intelligible basis, it is necessary to recognise the fact that *glycosuria may result from a surgical lesion.* We may consequently divide our cases into—

(1) Those in which the lesion demanding surgical aid is the actual and direct cause of the appearance of sugar in the urine.

(2) Those in which the sugar in the urine precedes, and is not dependent on the surgical lesion.

(*a*) The lesion and the glycosuria are independent conditions.

(*b*) The lesion is dependent on some previous glycosuric condition—gangrene, balanitis, &c.

Therefore, upon discovery of sugar in the urine, it is necessary to determine whether the patient has previously had distinct symptoms of diabetes, or whether the symptoms from which he is at present suffering are dependent upon the surgical malady or injury for which he is seeking advice.

In this way we shall have an opportunity of ascertaining whether a secondary glycosuria may influence the course of the malady or injury which has set it going, or render operations for the disease or injury liable to complications. That is, we shall have a chance of seeing whether, as Verneuil

and Tuffier believe, the presence alone of sugar in the urine is to be taken as indicating a reserved prognosis after operative proceedings.

We shall next consider those cases where the glycosuria has preceded the lesion for which the patient is seeking advice. And here we have two varieties of cases: those in which the lesion is due to the previous glycosuria—balanitis, carbuncle, gangrene; and those in which the lesion and the glycosuria are independent conditions. We shall have to consider in these cases the indications for, and results of, operations, and how far the surgeon ought to be influenced by the classical motto of the diabetic patient, "Noli me tangere."

At the present time diabetes is considered to be a term including more than one morbid condition. Lancereaux, for instance, recognises three clinical varieties:

(1) The lean variety.
(2) The fat variety.
(3) The traumatic variety.

This raises a question whether the effects of diabetes, from the surgical point of view, arise simply from the disturbance of the sugar metabolism, or whether they vary according to the nature of the prime cause of that disturbance, or for other reasons that we are not aware of.

This question, in the present state of medical science, it would be rash to answer with any degree of conclusiveness. But it appears that the majority of cases that come under the surgeon's care belong to the fat variety (*diabète gras*); also that diabetes resulting from traumatism seems but little predisposed to septic infection; and that in the thin variety septic infection is also rare, perhaps because these latter cases are rapid in evolution, and tissue-changes have not had time to assume that importance that they ultimately adopt in the more chronic and stout varieties.

## Part II.

### *Cases in which the Glycosuria is secondary to the Lesion for which Patient is seeking advice.*

These forms of glycosuria may, from the point of view of the surgeon, be conveniently divided according to their mode of causation into—

(1) *Toxic glycosuria.*

(A) *A septic form,* in which the sugar excretion is determined by the influence of micro-organisms upon the system, or upon individual tissues, *e. g.* carbuncle, phlegmon.

(B) *A form due to drugs.*—As the result of a number of drugs—chloroform, chloral, opium, morphia, &c.—a sugar reducing substance has appeared in the urine. There is, however, reason to believe that this substance in many cases is not glucose, but glycuronic acid.

(2) *Traumatic glycosuria.*—The sugar secretion is the direct result of some mechanical injury, such as a blow on the head, or a blow on the abdomen.

(1) *Toxic glycosuria; septic.*—Our subdivisions of the septic glycosurias are perfectly arbitrary, and are adopted merely for convenience. We shall consider glycosuria as secondary to phlegmon, abscess, carbuncle, erysipelas, and gangrene. The true classification would depend on the germ causing the lesion; but we have not at present enough evidence to justify us in attempting such a division of cases. Whether glycosuria is more common after one species of organism than another we do not know. That it follows other specific diseases than those common in surgery is well known. It has been noted frequently after measles, pneumonia, scarlet fever, influenza, &c. (Bordier, Frerichs, Saundby). These we shall not further consider.

For a knowledge of the great frequency of transitory glycosuria in these cases we are indebted to Redard's paper on "Ephemeral Glycosuria in Surgical Affections," 'Rev. de Chirurg.,' 1886. He would appear to have found glycosuria very frequently during the course of these cases. Giovanni Nuzzi ('Il Morgagni,' October, 1889), however, has doubted these facts. He found glycosuria three times in 115 obser-

vations, and in one only of these was suppuration present; and he concludes that ephemeral glycosuria in surgical affections is an exceptional fact, depending less on the affection itself than on the special state of the individual.

We think, however, considering the positive evidence of Prout, Trélat, Prévost, Redard and others, that glycosuria is undoubtedly frequent in the specific inflammations common in surgery. This glycosuria is generally of short duration, lasting a few days or perhaps hours only (ephemeral as Redard calls it); but apparently in rare cases it may persist, the patient becoming markedly diabetic.

These cases show us how impossible it is to draw any line between cases of glycosuria of transitory nature and cases presenting all the evidence of marked diabetes. Such an attempt, moreover, would be beyond the scope of the present paper. What we insist on here is that glycosuria, accompanied or not with polyuria, albuminuria, &c., may, and frequently does, occur as the result of the lesion for which the patient is presenting himself, and by its presence may lead to a possible erroneous diagnosis of a past glycosuric condition, and thus influence prognosis. Another practical point, that specially interests us here, is whether the sugar is of ill omen for the future course of the patient, as regards both his local and general condition.

*Phlegmon and glycosuria.*—Sugar is met with very frequently in the course of the cellular and cellulo-cutaneous inflammations included under the term phlegmon. Redard has met with glycosuria almost invariably at some time or other during the course of these cases. The first instance, that we have come across, where a specific inflammatory condition appears to have given rise to glycosuria is a case described by Latham.

A man admitted in 1789 at St. Bartholomew's Hospital, under the care of Dr. Pitcairn, had been bitten by a rat on the hand between index and thumb. The hand and arm swelled considerably; boils and abscesses appeared not only on the arm of the patient, but also on other parts of the body. From this moment the health of the patient altered very perceptibly, and emaciation supervened. The urine pre-

sented diabetic characters, as much by its amount as by its quality. Sugar existed in it in large amount.

The diabetes that here ensued has been attributed to fright (by Fischer), but it seems to us more reasonable to ascribe it to the cellulitis. The case is certainly not beyond criticism, for nothing is stated about the previous history.

In the majority of cases the sugar lasts for two or three days, sometimes less (one day), sometimes more (four or five days).

The highest amount of sugar noted in these cases is 2·29 per cent. (Redard, Obs. xxviii). In a case that we have lately seen, it declined from ·4 to ·19 per cent. The highest sp. gr. noted is 1029.

Redard has pointed out several important points with regard to ephemeral glycosuria, attendant on traumatisms and inflammatory disorders. The sugar in many cases appears on a rise of temperature, and it is noticeable that the sugar follows upon the rise, and is not the cause of the rise.

In his observations this writer says the glycosuria was accompanied almost always by a passing polyuria, an increase in the urea, and in density.

Albuminuria of a transitory nature is not infrequent; out of fourteen cases of glycosuria, occurring during the course of cellulitis, it is noted as existing three times; and one case shows well what so often happens, that the albumen and sugar replace one another. In a case of phlegmon of the hand and forearm (Redard, obs. xvii) albumen was noted on February 1st. On February 3rd the albumen had disappeared and sugar had made its appearance.

*Prognosis.*—This has to be considered from two points of view. (1) Does the presence of sugar in the urine lead one to give a more reserved prognosis as regards the phlegmon?

(2) What is the probability that the glycosuria may become a confirmed habit?

(1) The sugar generally appears with an exacerbation of the symptoms, frequently accompanying an attendant rise of temperature. But it does not appear that the cases in which it makes its appearance are unusually severe. Out of fourteen cases collected by us, eleven recovered.

One of the fatal cases was an old woman who had had

kelotomy performed, and who, five days later, developed inflammation of the forearm.

Nor is there any evidence that the glycosuric condition has influenced secondarily the course of the cellulitis. In all cases the sugar accompanies or follows any exacerbation of symptoms, it never precedes them.

(2) As regards the second point, there is only one case, that of Latham's, already quoted, where it would seem that a confirmed glycosuric habit had been set up.

This solitary case, and those that we shall mention as following carbuncle, abscess, and traumatisms, should lead us to carefully examine the urine till the sugar has entirely and permanently disappeared.

In a case that has lately fallen under our own observation agar and potato cultivations were made, and in both cases pure growths of *Staphylococcus pyogenes aureus* resulted in the first generation.

*Case of spreading phlegmon due to* Staphylococcus pyogenes aureus; *glycosuria.*—Male, æt. 20, powerful, well built, heavy; has been indulging rather freely of late in alcohol.

August 16th, 1892.—Cut by glass over extensor surface of forearm near elbow; wound sewn up.

17th.—Rather inflamed.

18th.—Stitches removed, and hot boracic fomentations.

19th.—$CHCl_3$, and wound scraped out.

27th.—Œdema having spread towards external condyle, counter opening was made. Great improvement, so that patient was allowed to go out.

September 4th.—Returned with fresh inflammation; incision with $CHCl_3$.

7th.—Another incision rendered necessary, with $CHCl_3$.

13th.—Slight rigor, 103·4°. Hitherto patient did not feel ill; to-day he is not well. A. C. E. mixture given, and fresh incision made above and below elbow.

14th.—Urine tested for first time; sp. gr. 1024, contains sugar 2 gr. ad ℥j (·4 per cent.).

17th.—Extension of brawny swelling in arm and forearm. Urine still contains sugar (·19 per cent.).

19th.—Gas and ether again given, and several incisions made.

21st and 22nd.—General improvement. Sugar absent from the urine.

24th.—Trace of sugar; two fresh incisions made under gas and ether.

October 5th.—Went to the seaside.

14th.—Returned, and had a small incision made under anæsthetic. Unfortunately urine was not tested.

November 3rd.—All wounds healed.

January, 1893.—Is able to play football.

*Abscess and glycosuria.*—Prout seems to have noticed the association of abscess and glycosuria in certain cases; but it was Prévost who, in a thesis (1877), drew attention to the close connection that existed between sugar secretion and purulent conditions.

Redard, from whom we quote these observations regarding Prévost, says that this observer noted the presence of sugar in all cases of suppuration, and Redard does not hesitate to confirm this.

Prévost went so far as to diagnose pus, somewhere in the economy, on discovering sugar in the urine. Here are two cases of his:—A man who had burnt his fingers had subsequent lymphangitis and adenitis. Prévost examined his urine, and during the few days he lived discovered sugar, which made him suspect the presence of pus. At the autopsy, all the lymphatics were found full of pus.

In a second case, a man who had had rheumatism was suddenly seized with violent pains in the loins. Prévost examined the urine, and finding sugar, came to the conclusion that suppuration would soon follow. Some days later, a perinephric phlegmon was noted.

We now know that the suppuration is not a necessary factor in the causation of glycosuria. V. Jaksch's statement ('Clin. Diag. trans.,' 3rd ed., p. 358), that fresh pus always contains glycogen, and that traces of grape sugar are seldom wanting, is of some interest in this connection, as is also the observation of Baumann and Bämmler, that acetone is common in pus.

The following case is a very clear example of transitory glycosuria following an abscess of the margin of the anus, and the case is all the more valuable from the fact that

examinations of urine made one year and two and a half years subsequently yielded negative results.

*Abscess of the margin of the anus; transitory glycosuria* ('Archiv. Gén. de Méd.,' 1888, i, 474).—H. D—, æt. 54, always in good health; no syphilis; a heavy smoker. September, 1885, suffered from a slight febrile attack with joint pains; nothing abnormal in the urine; perfect health till July, 1886.

July 28th.—Pain round the anus; abscess the size of a large walnut found; fever. *Urine* examined next day. Amount, 2½ litres; glucose, 11·20 grammes per litre (1·12 per cent.).

August 1st.—Abscess opened; sugar in the urine.

12th.—Still sugar in the urine; wound healing.

18th.—Cicatrisation complete; no glucose.

Examinations made September 21st, 1886, and January 15th, 1888, showed no sugar.

In this case the sugar lasted two or three weeks. In a remarkable case, described by Dr. Imlach ('Brit. Med. Journ.,' 1885, ii, 61), entitled "A case of Diabetes Mellitus cured by Removal of the Uterine Appendages," in addition to glycosuria, other marked diabetic symptoms existed, and lasted for four or five months. We can scarcely resist the conclusion that these symptoms were set up by the suppurating Fallopian tubes. Although the pyosalpinx was not diagnosed till a month after the patient had been afflicted with thirst and polyuria, it had doubtless preceded these symptoms in point of time. What is unusual in this case is the high specific gravity of the urine, 1036, the thirst, the large amount of sugar, and extreme polyuria with wasting. But as soon as the suppurative foci were removed things began to mend, and a month afterwards the patient was perfectly well.

*A case of diabetes mellitus cured by removal of the uterine appendages* (Francis Imlach, M.D., 'Brit. Med. Journ.,' 85, ii, p. 61).—K. G—, widow, æt. 31. Pyosalpinx diagnosed on February 12th, 1885. Operation suggested.

February 22nd.—The patient stated that for a month she had been afflicted with insatiable thirst and sleeplessness; that the bowels were habitually constipated, and the urine greatly increased in quantity. On examination, *urine* sp. gr.

1036; no albumen; sugar with Fehling's solution. All thoughts of operation abandoned.

Patient put on diabetic diet and treatment. Urine became reduced (from an unknown quantity) to five or six pints per diem, but there was but little diminution in the sugar. On April 15th she passed 2560 grains in twenty-four hours. As she was becoming rapidly weaker and more emaciated, she was admitted into the hospital May 15th, where anti-diabetic treatment was strictly maintained.

May 16th.—1200 grains of sugar.

May 19th.—Uterine appendages removed. Right Fallopian tube distended with pus. Left tube contained only a little muco-pus. Patient recovered without a bad symptom, and with a surprisingly level temperature chart. She got out of bed on the ninth day, and left the hospital June 3rd. During the first twenty-four hours between six and eight ounces of urine were drawn off every six hours. Sp. gr. during first six hours was 1028, then fell to 1114, then rose to 1034.

| | | | Sugar. | Diet. |
|---|---|---|---|---|
| May 19 | Op. | | | |
| „ 20 | 17 oz. in 24 hours | 1034 | Present | Milk and soda. |
| „ 21 | 21 „ | 1030 | „ | Barley-water. |
| „ 22 | 16 „ | 1030 | „ | „ |
| „ 23 | 15 „ | 1032 | „ | Beef tea. |
| „ 24 | 13 „ | 1030 | „ | Rice pudding. |
| „ 25 | 16 „ | 1030 | „ | „ |
| „ 26 | 17 „ | 1030 | Absent | Ordinary fare. |
| „ 27 | 18 „ | 1028 | „ | „ |
| „ 28 | 22 „ | 1024 | „ | „ |
| „ 29 | 22 „ | 1020 | „ | „ |
| „ 30 | 24 „ | 1024 | „ | „ |
| „ 31 | 24 „ | 1022 | „ | „ |
| June 1 | 24 „ | 1020 | „ | „ |
| „ 2 | 18 „ | 1024 | „ | „ |

Sugar tested with Fehling's solution gradually diminished until May 26th, when it finally disappeared.

June 12th.—Urine normal; sp. gr. 1020.

June 19th.—No sugar; sp. gr. 1010. Strength is now almost completely restored.

But the glycosuric condition may persist, and a case is recorded by Frerichs (quoted by Saundby, 'Lectures on Diabetes,' p. 36) where diabetes followed an abscess connected with a tooth.

A somewhat similar case is given by Hull ('Phil. Med. News,' 1882, xli, 708), where symptoms of diabetes followed an abscess of the gall bladder; frequent micturition; urine ten pints daily, sp. gr. 1030; large amount of sugar; trace of albumen; polydipsia. These symptoms lasted at least two months, and then disappeared; and although the patient died three months later of what Hull supposed to be diabetic coma, the evidence is far from convincing, for the post-mortem was not complete, and none of the diabetic symptoms had recurred.

In these two cases (Imlach's and Hull's) we have seen as the result of a toxic condition, not only glycosuria and polyuria, symptoms more or less common under such circumstances, but also thirst, weakness, and wasting. In both cases the symptoms disappeared, rapidly, after removal of the exciting cause.

We have remarked before how impossible it is to draw a line between transient glycosuria and what is generally considered as diabetes, and these cases well illustrate the difficulty. Both of them are described by their authors as diabetes mellitus.

*Prognosis.*—Has this transitory glycosuric or diabetic condition affected in any way the subsequent course of the case? In Imlach's case it certainly did not. The patient recovered after an abdominal operation with extreme rapidity. And in Hull's case the fistula that persisted was certainly not dependent on the glycosuric condition. The three cases of Imlach, Frerichs, and Hull point very clearly to the possibility of what is generally a transitory and harmless condition, leading to a more permanent and severe affection. More or less related to the preceding cases is one described by Dr. F. Taylor ('Guy's Hosp. Rep.,' 1880–81, p. 197), where a patient became diabetic after a gonorrhœa with orchitis, and died in about five months' time from coma.

*Gonorrhœa; diabetes; urethral calculus; removal; coma; death.*—Charles P—, æt. 25, always had had good health till he developed gonorrhœa six months ago. He came as an out-patient six weeks ago with gonorrhœa and orchitis; as he got well the symptoms of diabetes—glycosuria, polyuria, and polydipsia—were noticed.

January 2nd.—Was passing five or six quarts daily, with much sugar. Admitted January 11th ; looked fairly healthy. Urine 1035 ; sugar, but no albumen.

February 3rd.—Intense lumbar pain on the left side for two days. During the month the patient gained seven pounds in weight. Urine decreased to two and a half and three pints.

March.—Weight decreased, and urine increased to four to five pints.

April 12th.—Calculus impacted in the urethra ; removed by Mr. Davies Colley.

17th.—Pain over lower part of the abdomen ; sickness.

20th.—Delirious at night ; is semi-comatose ; skin cold ; vomits. Temperature 97·3° ; pulse 66 ; respiration 28. At 5 p.m. he became comatose and died.

*Carbuncle and glycosuria.*—Carbuncle has long been known to be a complication not infrequent in the course of diabetes. On the other hand, glucose may appear for the first time in a patient affected with carbuncle. Prout recognised this fact (1840). He stated that diabetes accompanies very often, and, according to his own experience, always, carbuncles, boils, and certain abscesses. Cheselden, according to him, recognised it in the last century ; and he points out that it is a matter of great importance to surgeons, as they mostly have the treatment of these cases. In 1852 there appeared in the ' Dublin Med. Press ' (vol. xxvii, p. 266) two well-reported cases quoted without the author's name from ' The Provincial Journal.' We give the first case as an example :

John B—, æt. 66, a packer, admitted into the Manchester Royal Infirmary December 12th, 1851. At the time, he presented on examination a large carbuncle on the back of his neck. Tongue moderately clean, and pulse weak. Crucial incision made and poultices applied.

The patient stated, on the question being put to him, that he thought the quantity of urine was increased. Urine measured 5 pints, sp. gr. 1031. Boiled with caustic potash it produced the characteristic colour of diabetic sugar. Boiled with copper sulphate and caustic potash in excess it yielded immediately the red oxide. A portion was

put on one side for about forty-eight hours, and when examined by the microscope showed abundance of torulæ.

Treatment conducted on ordinary surgical principles. Patient recovered rapidly, and in a few days not a trace of sugar could be discovered in the urine. The sugar remained for sixteen days after he came under treatment, and continued steadily diminishing.

| | | | | | |
|---|---|---|---|---|---|
| December | 17 | . . . Urine | 4½ | pints . . . | Sp. gr. 1031. |
| „ | 21 | . . . „ | 4 | „ . . . | „ 1030. |
| „ | 25 | . . . „ | 4 | „ . . . | „ 1026. |
| „ | 29 | . . . „ | 3½ | „ . . . | „ 1028. |
| „ | 31 | . . Less than | 3 | „ . . . | „ 1024, no sugar. |

In 1861 Philipeaux and Vulpian recorded a case in the 'Gaz. hebd.'

Marchal de Calvi, however, in the same year called these facts in question. He had since 1852 been drawing the attention of the French to diabetes as a cause of gangrene. He thought that Prout had read his cases upside down. "When Prout found sugar in cases of gangrene, was it not rather that gangrene had developed in individuals passing sugar for a longer or shorter time . . .? They are not gangrenous patients who become diabetic; they are diabetics who become gangrenous" ('L'Union Méd.,' 1861, xi, 2 s., p. 168). Trélat, writing later in the 'Dict. des. Sc. méd.,' after reviewing the evidence, answers the question, "Can a non-diabetic patient become diabetic the day he is affected with carbuncle?" in the affirmative. Redard speaks of meeting with sugar frequently in the urine of patients with carbuncles. He gives details of one case only.

It seems not improbable that a considerable number of cases of carbuncle that have been attributed to diabetes should rather be considered as cases of carbuncle with secondary and transitory glycosuria, and this raises the question whether carbuncle is as frequent in diabetes as it is considered to be.

*Duration of the glycosuria.*—The sugar commonly persists in the urine from two or three days to two or three weeks, disappearing as a rule with the carbuncle, or before.

In a case that Denucé communicated to Trélat the glycosuria is stated to have persisted for six months, in spite of the cure of the carbuncle. The patient is specially stated to have been non-diabetic on admission.

We can find no case where the diabetic habit seems to have been permanently set up by a carbuncle. Such a case would of necessity require a very clear history.

*Amount of urine.*—The amount of urine is only noted in three cases. In two of them it measured five and five and a half pints; in the other it is stated that there was no polyuria, but this was before the sugar was discovered.

In one of the cases where the quantity was given (case quoted in the 'Dub. Med. Press,' 1852, xxvii, p. 266, from 'The Prov. Journ.'), the amount of urine and the sp. gr. steadily diminished as the carbuncle healed.

| | | | |
|---|---|---|---|
| December 17 | . . | Urine 4½ pints | . . . Sp. gr. 1031, sugar. |
| „ 21 | . . . | „ 4 „ | . . . „ 1030 |
| „ 25 | . . . | „ 4 „ | . . . „ 1026 |
| „ 29 | . . . | „ 3½ „ | . . . „ 1028 |
| „ 31 | . . | Less than 3 „ | . . . „ 1024, no sugar. |

The sugar lasted for sixteen days after the patient came under treatment, and then vanished.

*Amount of sugar.*—Sugar varied from a trace up to 6·45 per cent.

*Other symptoms.*—In a case that Wagner described ('Archiv f. path. Anat.,' Bd. xii, S. 401, 1857), during the recrudescence of a carbuncle there appeared for the first time an inextinguishable thirst, a continual want to micturate; urine very pale, 1029, containing 5 per cent. of glucose. Patient, who was an intelligent man, said he had never previously suffered from any of these symptoms.

*Prognosis.*—Of 16 cases which we have collected, 2 died. Both these fatal cases were described by Wagner ('Archiv f. path. Anat.,' Bd. xii, S. 401, 1857). They both apparently died from secondary infection of the system. Wagner inquired very carefully into their past histories, and could obtain no evidence of diabetes. In one case the onset of symptoms was quite sudden four days before death—inextinguishable

thirst, a continual want to pass water; urine, sp. gr. 1029, sugar 5 per cent. In the other case sugar was not looked for till two hours after death, when it was found in some urine.

Fourteen of the cases recovered, and the carbuncles do not seem to have been very severe. One case (Morin, 3rd case quoted by Redard) was complicated with erysipelas, and the sugar, which had once disappeared, again put in an appearance for a short time.

But we have to consider the possibility of a diabetic habit being set up. Although the longest case we have here is one of six months, in somewhat similar toxic glycosuria there is reason to believe that diabetes has started from an erysipelas or an abscess. This should point out the necessity of keeping a patient under observation till the sugar has disappeared.

There is, on the whole, not sufficient evidence to show that the presence of glycosuria has influenced the course of the malady in any way for the worse. The case of Wagner's, where death occurred a few days after the onset of acute diabetic symptoms, might be pointed to as an example of the pernicious effect of glycosuria. But against this we have all the other cases where no ill results seem to have appeared; and even in Wagner's case the evidence would seem to show that the glycosuria was dependent on the increased severity of the patient's condition, and was not the cause of it.

*Erysipelas and glycosuria.*—Redard states that in cases of erysipelas, especially when the erysipelas is of some gravity, glycosuria is almost constant, and he gives three cases in detail.

In the nine cases we have collected, there is no history of diabetes before the onset of the erysipelas. The urine examined gave negative results. Erysipelas supervenes on a cut or operation wound, or apparently idiopathically, and sugar appears in the urine. The amount of sugar follows very closely the intensity of the erysipelas. When it improves, the sugar lessens or disappears; when it extends, sugar increases or reappears. In three of the cases albumen also appeared as a transitory condition.

| Sex and age. | Reference. | Condition of patient. | History of the diabetes. | Medical treatment. | Lesion. | Operation. | Complications. | Final result. | Remarks. |
|---|---|---|---|---|---|---|---|---|---|
| M., 67 | Dr. F. Taylor, 'Guy's Hosp. Rep.,' 1880-1, xxv, p. 199; under Mr. Howse, 1879 | No previous history given; moderately wasted at time of death | Was passing a very large quantity of saccharine urine | — | Large carbuncle on back of neck | Carbuncle opened 21st | 23rd, restless night, delirious; 24th, better, P. 132, T. 100°; 25th, became comatose | Death on 26th | P.M.—Granular kidneys, atheromatous arteries; no gout made out. |
| M., 66 | Fischer, 'Deut. med. Woch.,' 1876, S. 158 | Powerful and well nourished | Urine 1035; 5% sugar | At first, in spite of carbolic acid treatment, inflammation made rapid strides, but in few weeks sugar fell to 0 and the carbuncle healed up | — | — | — | — | With any indiscretion in diet sugar appeared; a year later it rose to 5%; disappeared on carbolic acid treatment. |
| M., 54 | Fauconneau-Dufresne, 'Union-méd.,' 1858, p. 490 | Stout | Diabetes suspected for long while; excoriations and eschars on legs long in healing; for two years enormous appetite and thirst. Urine was not tested till after death, then found to contain sugar | — | Carbuncle of neck | Incisions made; later more incisions required, but deferred till next day; had been getting up every day | In the night profound and sudden change; he was found motionless, cold, and with rattles in his throat | Death | From suddenness of change in his condition it looked as if he had been carried off by the diabetes, and not by septic condition. |
| M., 39 | Pavy, 'Brit. Med. Journ.,' 1885, ii, p. 1049 | — | Diabetes of four weeks' date seen a week after onset | — | Carbuncle over parotid | — | — | Death | Carbuncle apparently determined a fatal result in a very acute case of diabetes. |
| M., 53 | Kraske, 'Centr. f. Chirurg.,' 1881, S. 545 | Apparently healthy and well nourished | Gave history of increase of urine and great thirst; urine, 4–5 litres; sugar, 4·16%; carbuncle on the neck few months ago. | Anti-diabetic diet | Enormous carbuncle, left side of head; of three weeks' history | Incisions down to the periosteum; wet carbolic dressings | Went on well; a fortnight later sugar 3·12%; later on there developed inflammation in the right biceps and left iliac fossa; abscesses formed; incision. | Died next day, after incisions for abscesses. | — |

Redard points out that the sugar generally appears when the temperature is raised. One of Bordier's cases is, however, a marked exception to this rule. A woman had had erysipelas of the face for six days; no sugar on November 6th; 7th, the erysipelas spreads, the fever falls, and sugar appears; 9th to 20th, condition the same. On the 20th the fever reappears; an abscess forms in the neck, and the sugar disappears ('Arch. Gén. de Méd.,' 1868, ii, p. 183).

*Amount of sugar.*—This varies from traces to 5 or 6 per cent. The sp. gr. is (when given) raised—1030 in two of Redard's cases. Urine does not seem to have been much increased, 1100 grammes being the most indicated.

*Duration.*—In all the cases but one, the sugar disappeared as the erysipelas improved. Saundby, however, describes a case where erysipelas apparently started marked diabetic symptoms.

*Diabetes mellitus, following erysipelas; loss of sexual desire; slight polyuria; slight thirst; no knee-jerks; no wasting* (Case 3, 'Lectures on Diabetes,' 1891, p. 35).—Mr. Paul R—, æt. 48, consulted me in March, 1888, complaining of diabetes, of which he had been aware for three or four months, after an attack of erysipelas supervening in his foot from a cut got by treading on broken glass. The polyuria and thirst were slight, and there was no wasting; his chief complaint was his loss of sexual desire, which seemed to depress him mentally, although he was unmarried. His knee-jerks were absent. The urine measured from four to five pints daily, sp. gr. 1029, loaded with sugar.

*Prognosis.*—This case is of some importance from the point of view of prognosis. It would appear that any of these toxic disorders capable of giving rise to a temporary glycosuria, may, very occasionally, lead to the development of a permanent diabetic habit.

We have seen that the sugar is entirely dependent on the erysipelas, diminishing as this latter improves, increasing as the erysipelas spreads.

Does the sugar react on the erysipelas, and influence its course for the worse? Although we are unable to answer dogmatically in the negative, we are strongly of opinion that it does not.

The increased amount of sugar in the blood must be very small, and considering its short duration, any influence that it might have on the tissues must be exceedingly slight.

In three of the cases death occurred. All three were operation cases, complicated with severe erysipelas. Two of them were supposed to be cases of latent diabetes, and are described as such by Tuffier, and the complications and death are attributed to the diabetes. There is no evidence whatever of this, and these cases form good examples of the fallacy likely to arise under similar circumstances. For instance, a case without any previous evidence or suspicion of diabetes is operated upon; erysipelas supervenes; the urine is tested and sugar found; and the unfortunate result of the case is laid at once at the door of diabetes.

Too much stress cannot be laid upon the error of this mode of reasoning. To begin with, in none of these cases is there reason to suppose that there was glycosuria previous to the erysipelas; consequently one cannot assume that in consequence of a glycosuric habit the individual has presented an increased liability to septic infection. Secondly, in the cases that died the erysipelas was described as being very severe, and there is no question that erysipelas may be equally severe in non-glycosuric patients, and lead to a fatal result.

Considering all these cases of septic infection with glycosuria, it will be noted that on the whole the results were good; recovery is the rule. In cases that give a previous history of glycosuria the result is very different; the likelihood of a fatal result is very great.

*Neoplasms and glycosuria.*—Redard (Congress of Surgery, 1885) makes the statement that he has seen a certain number of cases of epitheliomata of rapid growth, in which he has noted the presence of marked quantities of sugar at different times; and that glycosuria shows itself especially in operations for such growths.

Mr. Spencer ('Westminster Hospital Reports,' 1888) has also noted the presence of sugar as the result of rapidly extending malignant disease, apparently in connection with the foul condition of the surface of the growth.

We shall not here consider the question of abdominal growths which occasionally give rise to glycosuria. It appears that if cancer of the pancreas is to give rise to glycosuria, the whole of the pancreas must be destroyed or rendered functionless. Glycosuria has also been noted as occurring after malignant disease of the intestines.

Leaving these cases we come to those cases of growths admitting of operation that more nearly concern the surgeon.

Redard ('Rev. de Chir.,' 1886, obs. viii) gives a case where ephemeral glycosuria occurred in the course of an epithelioma of the tongue.

A patient of previously good health had an extensive epithelioma of the tongue. This had been submitted to extensive cauterisation, with the result of causing much inflammation of the tongue and buccal mucous membrane.

Examination of the urine gave sp. gr. 1020, amount 820 c. c. in twenty-four hours; sugar ·4 per cent.; albumen ·01 per cent.

The next day the amount was 725 c.c.; sp. gr. 1015; sugar ·18 per cent.; albumen ·012 per cent.

On the following days the albumen and sugar disappeared.

Mr. Spencer ('Westminster Hospital Reports,' 1888) gives a case of a man with an ulcerated malignant growth on the back of the hand, and glycosuria, which disappeared when local treatment of the growth was carried out.

We have notes of several other cases that seem to belong to this class:

Epitheliomatous ulceration of the tongue (Verneuil).

Epithelioma of the lower jaw, ulcerated; this had been cauterised several times (Verneuil).

Ulcerated cancer of the breast (Fischer).

Epithelioma of the palate (Verneuil).

What is noticeable about these cases is that they were all ulcerated or inflamed; two of them had been cauterised. We have come across no case where glycosuria was the result of external growth, and where there was no ulceration. The question consequently arises whether the glycosuria is due to the inflammation and not to the growth. Mr. Spencer's case would seem to show this: as soon as local treatment was adopted the sugar disappeared.

*Traumatism and glycosuria.*—Although a few cases had been recorded at the end of the last century and the early part of this, not much attention was paid to the connection between head injuries and glycosuria till after the experiments of C. Bernard about 1850. A series of important papers were then published on the clinical relations between traumatism and diabetes, *e.g.* by Goolden, 1854, Leudet, 1857, Fritz, 1857, Griesinger, 1859, Fischer, 1862. Lately Anna Bernstein-Kohan has, in a thesis presented to the Faculty of Paris, reconsidered these cases. Lancereaux[1] quotes her paper extensively. But although by all these authors the occasional occurrence of well-marked cases was noted, it was reserved for Redard (1885) to point out the great frequency with which sugar occurs in the urine as the result of head and spinal injuries. "If one looks carefully for sugar in the urine one finds it almost always, with or without albuminuria, most frequently ephemeral, lasting a few hours only, but it may become permanently established." Pietro ('Rev. Clin. et Thérap.,' 1888) confirms these facts. He found glycosuria constantly in all cases of traumatism examined by him. Nuzzi, however, only found glycosuria three times in 115 observations on thirty-nine patients. Bernstein-Kohan out of forty-five cases found twenty-five following traumatism of the head, of which ten were of the occiput and nape of neck; thirteen following traumatism of the vertebral column; seven following injuries in various parts of the body, for the most part falls on the loins and abdomen. Fischer collected twenty-two cases where injuries other than those to the head caused glycosuria. But if Redard be correct, these figures give no idea of the frequency with which ephemeral glycosuria lasting a few hours only occurs after traumatisms of all kinds. He has noted it frequently after apparently light wounds—in several cases after simple fracture. In a case of fractured radius there was ·63 per cent. glucose immediately after the accident, which disappeared in a few days.

He has often found it after severe operations attended with shock; glycosuria in these cases is, however, much less frequent than albuminuria.

[1] 'Clinique Médicale,' 1879—1892, Paris, 1892.

*Amputation of breast; ephemeral glycosuria.*—X—, æt. 38. Cancer of right breast for six months, with considerable glandular enlargement.

Amputation of breast, and removal of axillary glands. Before operation, the urine examined with care showed no sugar or albumen. In the first two days after operation, no sugar. Third day, *urine* 750 grammes in 24 hours, acid, 1029; urea, 22·11; sugar, 3·30 per 1000, no albumen. Temperature 38° C. Next day no sugar or albumen. Local general condition is excellent. There was no sugar subsequently and the wound healed without complication.

On examining the cases it appears that sugar may immediately follow on the traumatism, that it is generally ephemeral, but may last some months or may become permanent; again the sugar may make its appearance later on, and is then more insidious in origin, and calls for a more reserved prognosis.

Bernstein-Kohan found that in eleven out of thirty-four cases glycosuria followed immediately after the accident; in eleven twenty to twenty-five days later, and in twelve from one month to six years.

Such figures do not, however, include the slight cases described by Redard and Pietro. These cases would swell enormously the list of those following the accident immediately. Sugar has varied in amount from ·1 to 7 per cent., polyuria and polydipsia are not constant, and polyphagia is relatively rare. An interesting point in these cases is that boils and phlegmonous lesions have been very rarely noted. Griesinger is quoted by Lancereaux to have noted one case. If this is correct, and there seems no reason to doubt it, it does not appear that the mere presence of sugar is sufficient to cause the liability to the septic infections that some varieties, and notably the stout variety, are prone to.

*Prognosis.*—In the large majority of cases where the sugar directly follows the traumatism, it is of but short duration. Occasionally it may be more persistent, lasting some months. It appears that if it is going to clear up it generally does so within four or five months; out of fourteen cases of cure only once was "diabetes" prolonged for two years (Brouardel).

The later the onset of the glycosuria, the more grave the prognosis (Lancereaux, Bernstein-Kohan).

We have next to consider whether the presence of the sugar has influenced the course of the lesion causing it or the operation for its relief. We have already noted that there does not seem to be in these cases any liability to phlegmonous inflammation. How far this is true for the later stages of those cases that become permanently diabetic we cannot say. Taking two cases of operation that Redard quotes, where ephemeral glycosuria occurred as a complication:

Amputation of breast; glycosuria on third day after operation; case without complication (quoted above).

Female, æt. 31, good health previously; no sign of diabetes. Severe wound of forearm, dividing all the soft parts. Urine 210 grammes, acid, 1035; urea, 1·9 per cent.; sugar, ·8; no albumen. Amputation performed. Urine after amputation contained a small quantity of sugar. Next day ·6 of glucose, in the evening no sugar. No complications are mentioned as having occurred. When the patient went out, the urine was normal.

Apparently in these cases the presence of sugar had caused no complication.

In cases of the previously diabetic, fractures are very slow in healing, as we shall show later on.

There have been a few cases in Guy's where with fractures there was a transient glycosuric condition, which seemed to have no ill result upon the union.

*Fracture of left radius in lower third* (Redard).—Male, æt. 47. Always had good health. No history of diabetes.

December 11th, 1882.—Fractured lower end of one radius; marked difficulty in the movements of hand and forearm without very violent pain.

12th.—Owing to the absence of pain Redard examined the urine and found 700 c.c. in twenty-four hours, acid, 1032; urea, 3·3 per cent.; sugar, ·63 per cent.; no albumen.

Analysis of blood showed ·22 per 1000. A trace after meals for a few days. None found after the sixteenth. Patient was given glucose (dry plums, &c.), for meals without producing any sugar in urine.

30th.—Patient went out well; urine contained nothing abnormal.

In a second case a fracture of clavicle, where sugar, ·24 per cent. appeared, it is not noted that there was any delay in healing.

*Scalp wound; concussion; fracture of left femur; glycosuria.*—Male, æt. 33, admitted November 13th, 1889, into Cornelius Ward, under Mr. Davies Colley.

A cottage pianoforte fell on patient from a height of eight feet. He became unconscious. He was brought into the surgery in state of collapse and partly unconscious; pulse 60, feeble. Pupils react to light. Epistaxis, and spitting up of a little blood.

A scalp wound, on right side of median line, over crown of head, measuring one inch in length and going down to the bone.

Left femur broken at junction of upper and middle thirds.

Limb put in a Hodgen's splint. Diet farinaceous.

14th.—Temperature 99·2°. *Urine* 1030, dark straw colour, no albumen or blood; distinct traces of sugar.

15th.—Some bleeding from the nose, which patient puts down to his scratching the mucous membrane.

16th.—Patient passed three pints twelve ounces in the last twenty-four hours. Distinct traces of sugar. Sp. gr. 1024, acid.

19th.—*Urine,* sp. gr. 1022, dark straw-colour, acid, no albumen, contains a large amount of sugar. Middle diet. Temperature last night 101°; this morning 99·6°.

21st.—*Urine,* specific gravity 1020; large quantity of sugar. Temperature last night 99·6°.

22nd.—Scalp wound almost healed; temperature 98°, and urine 1020; still sugar.

Patient says he is not losing flesh, is not a great eater, nor does he take much fluid to drink. Tongue moist and flabby. Right pupil a little larger than the left.

26th.—Urine pale yellow; large deposit of urates; sugar present.

29th.—There is some dulness at right apex, and there is some blood mixed with the expectoration.

December 2nd.—Expectoration no longer contains blood.
7th.—Urine of normal colour ; sp. gr. 1012. *No sugar.*
21st.—Expectoration and slight hæmoptysis.
31st.—Hodgen's splint removed ; plaster bandage.
January 2nd.—Patient gets on to a couch.
11th.—Discharged on crutches.
Shortening ¼ inch.

*Conclusions.*—(1) Sugar is very common in the urine in appreciable amounts, as the result of toxic (septic) and traumatic lesions.

(2) There is no evidence to prove, and no reason to suppose, that as the result of this secondary glycosuric condition, either the lesions themselves or operations for those lesions are rendered more severe.

(3) We shall see later on that the previously diabetic are liable to various complications ; therefore we must not, on discovering sugar in the urine, condemn a patient as liable to these complications till a full inquiry has been made into his past history and present condition.

(4) We think, therefore, that we are justified in separating, as far as possible, those cases where the sugar is the result of the lesion for which the patient is seeking advice, from those where the lesion occurs in a patient previously glycosuric.

(5) There is no reason to suppose, as many following Verneuil do, that the presence of sugar in the urine necessarily implies a more guarded prognosis.

(6) On discovering sugar in the urine, if this be found to be secondary to the lesion demanding operative treatment, and not to a previously glycosuric habit, there is no contra-indication to operation ; and there is no evidence to show that the results will be any worse than in non-glycosuric patients.

## Part III.

### *Operations in diabetes.*

These may be divided into two main divisions :

(1) Operations that are urgent.

(2) Operations that are not of immediate necessity.

(1) *Urgent operations.*—As examples of these we may mention strangulated hernia, operations for hæmorrhage, or injuries. Under such circumstances some operations must be performed, and any complications dependent on the diabetes must be risked. It is obvious that the results of these operations are dependent on several factors—the condition of the patient, the nature of the lesion, and the gravity of operation required. The only rule to lay down in these cases is to perform that operation that involves least disturbance of parts and causes least shock to the patient. We have not enough cases to give any idea of the results of operations of urgency in diabetics, but the prognosis may be inferred from the general considerations that we shall mention under the second heading.

As an example of an operation of urgency we may quote a case quoted by Dr. Ashurst ('Phil. Med. News,' 1888, vol. liii, p. 705).

A man of middle age had long had diabetes. He came under Dr. Ashurst's care with strangulated hernia. Upon operation a small patch of gangrene was found on bowel, which was left in the wound after division of constriction. The liability of diabetics to infection by septic material was shown the next day, when not only the bowel but the edges of the wound and the surrounding tissues were gangrenous.

Verneuil did not hesitate to propose an operation in the following case (Tuffier, 'Archiv. Gén. de Méd.,' 1888, vol. ii, obs. vii).

A man of fifty, thin and pale, diabetic for ten years (5 per cent. of sugar) ; in spite of treatment sugar has never fallen below 2 per cent. For a year there had been sudden and violent rectal hæmorrhages ; symptoms of obstruction, constipation, distended abdomen ; epithelioma of rectum diagnosed. On account of severe pain, hæmorrhage, and threatening

obstruction, Verneuil advised inguinal colotomy as involving the least disturbance of parts.

(2) *Operations that are not immediately called for.*—This heading includes all operations that would under ordinary circumstances be performed. The indications in these cases depend on (*a*) the condition of the patient, on (*b*) the nature of the disease, and on (*c*) the severity of the operation required. We shall consider these indications at some length.

(*a*) *The condition of the patient.*—Here we shall have to consider the degree of diabetic cachexia and the duration of the diabetes; the amount of sugar and the results of treatment in diminishing the quantity; the presence or absence of concomitant cachexia; the prognostic significance of the knee-jerk.

*Variety of diabetes.*—Most of the cases that come under the surgeon's notice appear to belong to the stout class of diabetics. It will be seen on looking through our cases that most of them are described as healthy-looking, vigorous, and robust; and in a considerable number it is specially noted that the patients were stout.

Tuffier remarks the same thing with regard to his cases: "The greater number of our cases had not arrived at the period of organic decay which marks the diabetic cachexia, and with the exception of one or two cases they all belonged to the category of 'diabète gras.'"

Verneuil again says, "It is said that fat diabetics are less exposed than thin. Well, I have seen succumb to carbuncle, gangrene, and diffuse inflammation, more fat diabetics than thin." And this statement is supported by Lancereaux, who says he has only seen two cases of furuncle and carbuncle in thin diabetics, perhaps, he thinks, because the rapid progress of these cases does not favour their evolution. And perhaps it is this rapid progress in the thin diabetics that prevents them coming under the surgeon's observation very often.

Most of the cases, of which we have notes, were apparently of good health, in many cases not very much affected by the diabetes, which had been recognised for some years. And it is obvious that the longer a case lives, the more likelihood there will be of certain structural changes taking place in

the system, leading to a lessened power of resistance to sepsis, and diminished power of repair after injury.

According to Lancereaux it is mainly at the age of forty-five to fifty that symptoms first become noticeable, although glycosuria may have existed for many years previously.

*Age of patients.*—The majority of the patients are above the age of forty-five ;

| | |
|---|---|
| Below 20 years | 2 cases. |
| From 20—30 years | 0 „ |
| „ 30—40 „ | 5 „ |
| „ 40—50 „ | 4 „ |
| „ 50—60 „ | 9 „ |
| „ 60—70 „ | 9 „ |
| Above 70 „ | 1 case. |

and these figures leave out the cases of gangrene occurring in diabetes, which would further swell the numbers at the later period of life. The large number of cases occurring after forty as compared with those before, is partly due to the greater number of cases of diabetes occurring at this time of life (from forty to sixty, according to Pavy and Schmitz, the largest number of cases is found) ; partly to the fact that many of the cases are malignant growths that occur towards the close of the term of life ; and partly that many of the lesions probably occur at a late period of the diabetes, and are secondary to it—*e. g.* cataract, balano-posthitis, and gangrene.

*Concomitant cachexia.*—Of these the most important is alcohol. Unfortunately the thirst that diabetes entails frequently leads to excessive imbibition of alcohol. The results of operations and injuries in such patients are bad in the extreme. Verneuil has proposed to call these cases alcohol-diabetics, and gives several examples of them.

A man of fifty-five, alcoholic and diabetic, pinched his finger. Gangrene of the finger and inflammation of the hand and forearm ensued, leading to death in a month after the accident.

A man of fifty-four, alcoholic and diabetic, bruised his tibia. There was subsequent sloughing of the soft parts over the tibia, and extension of inflammation to the thigh.

Death occurred seven weeks after the accident, delirium, followed by coma, preceding it ('Gaz. hebd.,' 1877, p. 664).

A man of sixty-two, diabetic and alcoholic, with an epithelioma of palate; 7·6 per cent. sugar. Sugar disappeared on treatment. Cheek incised; growth removed by thermocautery. Hæmorrhage two hours after operation; the next day pneumonia started, followed by delirium, coma, and death ('Bull. Soc. de Chir.,' 1884, p. 378).

*The amount of sugar and the results of treatment in diminishing the amount.*—What indications are to be drawn by the surgeon desiring to operate from the presence or not of sugar in the urine, and from the result of treatment in causing the sugar to diminish or disappear?

With regard to the results of preparatory treatment Verneuil and Tuffier are dogmatic. "If medical treatment," says Tuffier, "carefully carried out reduces the amount of sugar, and especially if it causes it to disappear, operate . . . . If medical treatment is inefficacious, one ought not to operate."

We will first consider—

*Cases in which preparatory treatment has been carried out.*—On examining the cases of growth on which operations were performed, it will be seen that the cases may be arranged under three headings.

(*a*) Cases in which sugar disappeared and good results followed.

(*b*) Cases in which complications and death ensued in spite of the disappearance of sugar before operation.

(*c*) Cases in which good results followed in spite of the presence of large amounts of sugar.

Of eight cases of operation in which sugar is stated to have disappeared as the result of treatment, or to have been absent before operation, three died. In one of these there had been no sugar noted for two years, and nevertheless after operation the sugar returned, and the patient died apparently of coma (No. 12). This and the following numbers refer to the tabulated cases on p. 368 *et seq.* In the other two cases sugar also returned the day after operation, one case dying as the result of erysipelas, the other of coma (1 and 11).

As opposed to these, in certain cases where in spite of

treatment the sugar remained in considerable amount (5, 7, and 8) 4·6 per cent., 3·3 per cent., 1 per cent., recovery took place without complication.

These cases are sufficient to show that we are not able to rely implicitly on the amount of sugar present before operation, for, as we have seen, in spite of its absence two cases died of coma.

Nevertheless we must not conclude that previous medical treatment has not influenced some of the cases for the better. Of thirteen cases in which it is distinctly stated that treatment was adopted beforehand only three died, and two of them were severe operations (11, 12).

We have not enough cases where preparatory treatment was not carried out beforehand to compare with these. But what cases there are would seem to indicate a bad result.

*Cases in which no preparatory treatment was carried out.*—We have six cases in which sugar was not found till after the operation, when a distinct history of diabetes was obtained. In these cases no preparatory treatment was undertaken; three of them died, two from coma, one from gangrenous complications, and of the three that recovered, one narrowly escaped death, severe inflammatory and gangrenous complication following the operation of circumcision for diabetic balanitis and phimosis.

We must remember, however, that in four of these cases it was only the supervention of severe complications that led to an examination of the urine and to the finding of a diabetic history. If the operation wounds had healed without further trouble, the diabetes would probably never have been discovered. What proportion of cases have in this way escaped detection we do not know; consequently any attempt to draw accurate conclusions would be impossible.

*Cases in which operation was refused on account of the non-diminution of sugar.*—We have already quoted Tuffier to the effect that "if medical treatment is inefficacious do not operate." Our cases certainly do not lead us to support this view in the slightest; as we have already pointed out, in many cases when sugar disappears death results; in

many others when it is persistent recovery without complication ensues.

Tuffier proceeds to give a series of cases where the above dictum has been acted upon. One case was a parotid tumour of slow growth in an old diabetic patient, which was not operated upon, and perhaps with perfect justice. But with regard to the other three cases, the masterly inactivity that was displayed was entirely uncalled for.

A tumour of breast of two years' growth (malignant) in a lady markedly stout and of *healthy appearance.* Sugar persisted in spite of treatment. No operation. Death a year later by generalisation in the lungs (obs. xiii).

Scirrhus of breast of three years' date in a woman of sixty-four; glands slightly enlarged; *general condition excellent.* Refusal to operate (Juteau, obs. xiv).

Osteo-sarcoma of upper maxilla in a diabetic; seventy-nine grammes of sugar a day. Patient had been to Vichy four years previously. Operation refused by Verneuil (obs. xv).

In these cases, more especially the first two, it would surely have been better to run the risk of an operation rather than let the patients suffer the miserable death that malignant growth entails. Moreover in the two cases of cancer of breast strict asepsis could have been maintained.

We agree with Landau in this matter, that in cancer admitting of operation we have to remember that diabetes may last ten years or more in old or stout people; on the other hand, carcinoma usually leads rapidly to death. Therefore we ought not to be frightened at energetic measures. There is no doubt the danger is greater in diabetes, but where "there is everything to lose one must dare much."

*Conclusions.*—(1) As far as the evidence goes it points to the beneficial effect of previous treatment. This probably produces its effect by lessening the tendency to coma. It is evident that it cannot alter those structural changes of vessels and tissues, &c., which have already occurred, and which are largely responsible for the ill results that occur.

(2) But although the sugar disappears, not only does the liability to septic infection remain, but the patient may also be carried off in a few cases by coma.

(3) We must not be deterred from operation merely because

the sugar does not diminish. In these cases we must consider not only the general condition of the patient, but the nature of the disease, and the operation required.

*Condition of reflexes.*—Absence, first noted by Bouchard (1881) (Saundby, p. 109) in nineteen out of sixty cases, or in 29 per cent. Of those in which it was absent 31·5 per cent. died (6), while of others only 2 or 4·2 per cent. died, which suggests that it is of bad prognosis. The jerk may reappear, if the symptoms improve (Marie and Guinon). Rosenstein does not agree in regarding the loss as of grave importance, as he found that it bears no relation to the amount of sugar, or to the acetone or ferric chloride reaction-giving substances in urine.

Reynier ('Bull. Soc. de Chir.,' 1887, p. 444) thinks the knee-jerk is of great importance from a prognostic point of view. The surgeon ought to examine with care the reflexes, and principally the patellar reflex. The absence of the reflex ought to prevent him from touching the patient, unless from absolute necessity, and ought to make him give a grave prognosis. Its presence will cause him a little less fear, whilst in cases of diminution he ought to wait and give a guarded prognosis. He bases this opinion on certain cases which have been under his care.

He divides them into three classes.

(1) *Those in which the patellar reflex was present.*

Here he had two cases:

(*a*) Case of sebaceous cyst, in which suppuration followed operation, and healing did not occur for fifteen days (15).[1]

(*b*) Epithelioma of lip, where again healing was delayed (16), and cicatrisation was not complete for a month.

(2) *Cases in which the patellar reflex was diminished.*

Here the prognosis is somewhat worse, depending on the return or not of the patellar reflex.

Case of a man, æt. 64, with carbuncle. On admission 145 grammes of sugar in the twenty-four hours. Diminution of reflexes. Incisions with thermo-cautery:—

[1] *Vide* Table of Cases, p. 368, &c.

| | | | | |
|---|---|---|---|---|
| | 4th day | Sugar | 155 gr. | Reflex almost disappeared. |
| | 6th „ | „ | 180 „ | |
| Under régime | 9th „ | „ | 120 „ | Gradual reappearance of patellar reflex. |
| „ | 11th „ | „ | 70 „ | |
| | | | 50 „ | |
| | | | 15 „ | |
| | | | 10 „ | |
| | | Sugar disappeared | | |

Patient was cured and cicatrisation was complete in two and a half months. Here there is a marked parallelism between the amount of sugar and the patellar reflex.

Increase of sugar, diminution of reflex; diminution of sugar, return of reflex.

(3) *Cases in which the patellar reflex was abolished.*

Here four cases are given, all fatal:

(*a*) Gangrenous complications after a wound.

(*b*) Lymphangeitis following wound of a corn.

(*c*) Phlegmon of the thumb following a prick.

(*d*) Gangrene of the great toe.

On examining these cases it will be at once evident that an examination of the reflex was scarcely needed to aid in the prognosis.

The cases that recovered were two very slight operations, one of which ought to have been kept aseptic. We doubt whether the presence or absence of the patellar reflex would have influenced a surgeon much as regards operation. The other cases in which the patellar reflex was absent are very different indeed, and cannot be compared with the first two.

In these cases the presence or not of the reflex was not of much help. They were all accidents of an inflammatory and gangrenous nature that are notoriously fatal in diabetics.

For purposes of fair comparison we ought to have had in the second and third divisions simple cases, such as were given in the first division, otherwise conclusions must be erroneous and useless.

Consequently more observations are required, though we very much doubt the utility of the symptom. It seems to us to be entirely secondary to the general and local con-

ditions which indicate far more exactly the gravity of these cases.

It is of some interest to note the parallelism between the diminution of the patellar reflex with the increase in the amount of sugar (especially noted in the case under the second heading above). It is not, however, peculiar to diabetics, for Berger gives a case where in an albuminuric patient the disappearance of the reflex coincided with the appearance of a local complication of a gangrenous nature, and a general state of great gravity. The reflex reappeared as the result of local amelioration and cessation of the grave general condition.

We think, therefore, that more evidence is required before the condition of the reflex can be considered to be of any prognostic significance.

*The nature and site of the disease, and the gravity of the operation demanded.*—We have considered what influence the condition of the patient has on the surgeon with regard to operation. We now consider the influence that the disease itself and the operation required ought to exert. Quite as important as the gravity of the operation required is the seat of the operation, and the possibility of keeping the wound aseptic.

For instance, circumcision in a healthy individual is an operation with next to no risks; but in a diabetic it is fraught with considerable danger. It is impossible to consider all the cases together. It is of importance, as far as possible, to separate wounds that may be kept aseptic from those where it is impossible.

We shall first consider the question of operation for neoplasms.

### *Operations on Neoplasms.*

We have collected eighteen cases of growth which were operated upon. In two of these the diabetes was not noted till after operation, and then only on account of complications —hæmorrhage and gangrene.

| No. | Age and sex. | Reference. | Condition of patient. | History of the diabetes. | Medical treatment before operation or not. | Lesion. | Operation. | Complications. | Final result. | Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|
| 1 | M., 45 | Verneuil, 'Bull. Soc. Chir.,'1878, p. 474, and Tuffier, loc. cit., obs. i | Big, strong, of healthy appearance | 2 months previously glycosuric; sugar 30 grms. per litre; cured by treatment | No sugar at time of operation; previous treatment | Parotid tumour, recently rapid in growth | Long and laborious operation | Next day much sugar; disappeared on following day; 4th day erysipelas; hæmorrhage on 6th, 7th, and 8th days | Death from hæmorrhage from ulcerated carotid | Recurrence of glycosuria after operation; ? whether bad results were due to diabetes |
| 2 | F., 56 | Obs. de Verneuil; Tuffier, loc. cit., obs. ix | Healthy looking | Recognised as glycosuric 3 years previously; urine, when patient was seen, contained sugar 4 grms. per litre | Medical treatment from Oct., 1879, to Jan., 1880; not noted whether sugar disappeared | Scirrhus of breast first noticed Aug., 1879 | Amputation of breast Jan. 19th | No complications; patient was up on 11th day; 4 days later urine examined, 3 grms. sugar per litre | Recovery | — |
| | | | | | | Return in axillary glands | Removed July 19th, 1880 | Hæmorrhage an hour after operation and on 7th day | Recovery | — |
| 3 | M., 64 | Tuffier, obs. xix | Apparently good health, marked stoutness, muscular strength slight | Intermittent glycosuria for 2 years; 4 grms. per 1000 when seen | Medical treatment for 15 days; complete disappearance of sugar | Epithelioma of tongue and pillar of fauces | $CHCl_3$; removed with thermo-cautery | Sugar reappeared in urine, 2—3 grms. per 1000; no complications | Recovery | Recurrence of glycosuria after operation. |
| 4 | M., 52 | Obs. de Verneuil; Tuffier, obs. viii | Vigorous, active, apparently good health | 4 years ago wasting; glycosuria found; disappearing as result of treatment; intermittent diabetes | No sugar at time of operation; previous treatment | Epithelioma of tongue, grafted on psoriasis | Removed by thermo-cautery | Sugar reappeared next day; tongue swollen; slough separated 7th day. Went out on 19th day with 8 grms. | Recovery | Recurrence of glycosuria after operation. |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| 5 | M., 50 | Verneuil; Tuffier, obs. xxii | Tubercular infiltration, summit of left lung | Diabetic for 7 years; several times under treatment; urine 2·750 litres a day; sugar 46 grms. per 1000 | Treatment; sugar not diminished | Epithelioma, soft palate and tonsil | Cauterisations with chromic acid | | oma said to be cured; death 2 mos. later of phthisis | diminished by treatment; good result. |
| 6 | F., | Berger, 'Bull. Soc. de Chir.,' 1883, p. 369 | — | Severe diabetes for over 10 years; 20—30 grms. per 1000 | Operation deferred till entire disappearance of sugar | Encephaloid cancer of left breast, of rapid growth | Amputation of breast, and removal of axillary glands | No reappearance of sugar; greater part of wound healed by first intention | Recovery | — |
| 7 | M., 65 | Richelot, 'Bull. Soc. de Chir.,' 1883, p. 374 | Vigorous, active; appears endowed with robust health; inveterate malaria | Old diabetes; carbuncle 7 years ago | Treatment; urine fell from 3 to 1½ litres; sugar from 40 to 33 grms. per 1000 | Epithelioma, inner surface of cheek | Removal of epithelioma and a submaxillary gland | Wound healed by first intention; small collection of pus in cervical wound, let out; well next day | Recovery | Practically no complication, in spite of large amount of sugar and malaria. |
| 8 | M., 47 | Redard, 'Rev. de Chirurg.,' 1886, Obs. vi, p. 503 | — | For 2 years rapid loss of strength, polydipsia, polyuria, troubles of vision; sugar, when first seen, 37·7 grms. per 1000; treatment; sugar diminishing to 20 grms., and at times to 7 grms., 6 grms., 1·7 grms. | Treatment for a few days; amount of sugar remained at about 10 grms. | Epulis; recent rapid increase in size (myxosarcoma) | Removal by thermocautery without anæsthetic | Healing without complications; sugar increased for 6 days from 10 to 25·2 grms., then diminished to 12 by 15th day | Recovery | Increase of sugar as result of operation. |
| 9 | M., 50 | Verneuil, 'Bull. de l'Acad. de Méd.,' 1881, t. x, 1471 | Big, strong, appearance of robust health; had malaria | Diabetes; sugar sometimes almost vanishes; glucose about 12 grms. a day | Treatment for 15 days; sugar disappeared | Epithelioma of penis | Amputation with galvanocaustic loop | Wound healed well without complications; intermittent fever on following day; sugar reappeared on 15th day | Recovery | Reappearance of sugar; intermittent fever. |

| No. | Age and sex. | Reference. | Condition of patient. | History of the diabetes. | Medical treatment before operation or not. | Lesion. | Operation. | Complications. | Final result. | Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|
| 10 | M., 50 | Verneuil, loc. cit. | Tall, muscular, still stout; strength does not correspond to appearance; malarial | Has lost flesh and strength; large eater; drinks a great deal to slake pronounced thirst; urine 4 litres a day; contains 5·40 grms. of glucose, 78 urea | Anti-diabetic régime for 10 days; urine diminished to 2½ litres; sugar disappeared | Epithelioma of tongue, repeatedly cauterised; rapid progress; bleeding | Tongue removed with thermo-cautery | 6 days later considerable hæmorrhage; fœtid expectoration; ? fœtid bronchitis; urine subsequently returned to 3 litres; sugar was not found | Recovery | Re-appearance of polyuria; no sugar; hæmorrhage due to separation of sloughs. ? due to diabetes |
| 11 | F., 47 | Landau, 'Berlin. Wochenschr.,' Bd. 43, s. 863 | Stout | Treated for diabetes 12 months ago; sugar; thirst, loss of strength; strict diet and treatment; got well | No sugar found at time of operation | Carcinoma, cervix uteri | Operation by Richelot's method | 23 hours after, dyspnœa and coma; urine became less, and contained sugar | Died 41 hours after operation; coma | In spite of absence of sugar at the time of operation; recurrence of sugar. |
| 12 | M., 60 | Hoffa, 'Münch. med. Wochen.,' 1888, s. 888 | Hitherto no want of health; at present very corpulent; liver large; urine contains trace of albumen; no sugar | 5 years ago diabetes and albuminuria; thrice visited Carlsbad, resulting in stoppage of glycosuria each time; for 2 years no sugar found | No sugar for 2 years (no treatment mentioned) | Carcinoma of rectum; abdominal pain and scanty fæces in 1885; blood in 1887 | Operation June 8th, 1888, by Kraske's method; no excessive bleeding; wound plugged with $CHI_3$; came round well after operation; anæsthetic well taken; Tr. Opii ♏x | Evening of day of operation 2·4 per cent. of sugar. 9th.—Restless; ? iodine poisoning; no sugar; left off $CHI_3$ dressing. 12th.—Iodine in urine less; return of ·2 per cent. sugar. 13th.—Hardly trace of iodine; 4·0 per cent. sugar. 4 days coma | Death after 4 days coma; ? cause | Re-appearance of sugar after 2 years' absence, and death from coma; ? cause. Antagonism of $CHI_3$ and diabetes. ? Bad kidneys and albuminuria and opium. |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | 61 | berger, W. Müller, 'Aerztlich Intell.,' 1882, s. 457 | | 6 years; thirst, &c.; 3 years ago went to Carlsbad; sugar diminished, but did not disappear | | breast; ? 2 years' history; first painless, then with pain; no glanular enlargement | with strictest care on 4th | union. 8th.—Considered well. 14th.—Hot skin, dry tongue; corner where drainage-tube was found unhealed; pyrexia and delirium followed sepsis | Death after 12 hours' unconsciousness | These two cases show that it is not sufficient to get rid of sugar, as Verneuil, Tuffier, and Roser think. |
| 14 | M., 62 | Jacobson; admitted Guy's Hospital Aug. 11th, 1891, Rep. Surgical Wards | — | Patient had diabetes for 18 months, 10 years ago. Urine now dark, with sediment, 1018, alkaline, contains albumen and sugar | Slight diminution of sugar by diet and codeia | Extensive epithelioma of penis; glands enlarged in both groins | Very extensive extirpation of penis, both corp. cavernosa and their crura, and last 3 in. of corp. spong., being removed Aug. 15th, chloroform and ether | Aug. 16th.—Patient lying prostrate; abdomen distended; wound looking well. Towards the close the wound sloughed, and the left foot became gangrenous | Death Aug. 25th, 10 days after operation | Patient never became himself after the operation: at times comatose, at others restless. |
| 15 | — | Reynier, 'Bull. Soc. de Chir.,' 1887, t. xiii, 444 | Good health up till now | At time of operation sugar did not exceed 10—15 grms. | — | Sebaceous cyst, cheek | Operation | Local inflammation and suppuration; cicatrisation took 15 days | Recovery | Healing delayed. |
| 16 | — | Reynier, loc. cit. | Good health formerly | Sugar formerly 60 grms.; at time of operation did not exceed 10—15 grms. | — | Epithelioma, lower lip | Operation | Wound partly broke down; cicatrisation complete in a month | Recovery | Healing delayed. |

*Cases of Growth in which Sugar was found for first time* after Operation, *and in which* after *Operation a history of Diabetes was obtained.*

| No. | Age and sex. | Reference. | Lesion. | Operation. | Complications. | History of the diabetes. | Final result. |
|---|---|---|---|---|---|---|---|
| 17 | M., 62 | Polaillon, Tuffier, loc. cit., obs. v | Parotid tumour for some months | July 12th.—Parotid tumour removed; operation rapid; no difficulty; chloroform | Prolonged primary hæmorrhage with, however, but slight loss of blood. Patient became drowsy, unaccounted for by loss of blood, which was unimportant | July 13th.—Pulse small; frequent delirium; urine examined, contained sugar; coma | 14th.—Death; diabetic coma. |
| 18 | F., 64 | Mannoury, 'Bull. Soc. de Chir.,' 1879, p. 755, obs. i | Epithelioma of mouth | Sept. 6th.—Removal after slitting cheek with bistoury and thermo-cautery under $CHCl_3$ | Wound looked well till 9th, when gangrenous patch developed. 10th.—Rapid progress of gangrene. Distinct history of diabetes during last 3 years; thirst, polyuria, whitlow, &c. | Examination of urine showed 80 grms. glucose per litre | 10th.—Death at 5 p.m. |

The remaining sixteen cases comprised :—

| | Recovered. | Death. |
|---|---|---|
| 1 parotid tumour, recently of rapid growth | — | 1 |
| 3 carcinomata of breast | 2 | 1 |
| 6 cases, malignant growth of buccal cavity | 6 | — |
| 2 cases, epithelioma of penis | 1 | 1 |
| 1 case, carcinoma of cervix uteri | — | 1 |
| 1 case, carcinoma of rectum | — | 1 |
| 1 case, epithelioma of lip | 1 | — |
| 1 case, sebaceous tumour of cheek | 1 | — |
| | 11 | 5 |

(1) Of these cases there were seven wounds where strict asepsis would have been possible; of these, however, four became infected and two died, one of erysipelas, the other through infection by some pyogenic organism.

(2) Out of ten operation wounds, where strict asepsis was not possible, three died, and of these every one (11, 12, and 14) showed evidence of coma before death.

The three latter cases of death could probably not be prevented, and it will always be most difficult even by the strictest means of asepsis to prevent death by infection in wounds where the patient is comatose, as in these three cases of death. In one of these cases, Rosenberger's, the strictest care was maintained during operation, and apparently for some days later. Then, apparently, the strict measures were relaxed, and infection took place through the drainage-tube opening.

The question of infection and death from this cause evidently does not depend on the gravity of the operation.

The question of coma is more difficult to decide. Certain cases suffering from severe infective complications, carbuncle, gangrene, and phlegmons, apparently have their end brought about partly by poisoning from the septic focus, partly by toxines due to the diabetes itself. These we shall not consider further, the sepsis being the main exciting factor.

If we except a few cases where coma resulted in young people from such a slight operation as cataract, it appears that coma followed somewhat severe and prolonged operations. In one case (11) Richelot's operation for carcinoma cervicis uteri; in another case (12) Kraske's method of

excision of rectum. The ages of the patients were respectively forty-seven and sixty.

Thus it appears from the scanty material at our disposal that coma directly produced by the shock of operation, whilst occurring in quite young people as the result of trivial operations, requires in elder people a severe and prolonged operation.

### *Operations for Cataract in Diabetics.*

Opinion is generally in favour of operation for cataract in diabetes, and the results are usually good.

We give details of a few cases to show, however, that there is some slight risk.

That this risk is small is shown by a case described by Thomas, where a woman in the last stage of diabetes was operated upon with success. The wound healed by the fourth day. She then began to worry about her husband not coming to fetch her; and, as the result apparently of this worry, became comatose and died.

It does not appear, if careful antiseptic precautions are taken, that the risk of inflammation of the orbital contents is very great.

Morton (1888) gives a case where a young girl was operated upon for cataract (needle operation). The orbital contents sloughed and became gangrenous, and the patient became comatose on the third day and died.

A child of fourteen (Guersant) died suddenly on the day of operation, without anything but the diabetes to account for the death.

The first case of Panas shows that the entire absence of sugar is not necessary to a successful result. The left eye was operated upon, when, in spite of treatment, sugar was still 15 grs. in twenty-four hours; and a second case of his had 3·60 per 1000 when operated upon. He seems to have had very successful results in the reduction of sugar from using antipyrine.

### *Operation for Balano-posthitis.*

The dangers of circumcision for balano-posthitis and phimosis in diabetic patients have often been pointed out.

Fournier ('Gaz. Méd. de Paris,' 1887, iv, 133) says nothing can be more grave than this operation in a diabetic patient, "it may cost him his life." M. Dubuc ('Gaz. des Hôp.,' 1874, p. 803) and M. Reliquet ('Gaz. des Hôp.,' 1874, p. 814) refused to operate for phimosis in diabetic patients. Non-intervention was supported also by Verneuil. Both Fournier and De Beauvais ('Gaz. des Hôp.,' 1874, p. 876) say that circumcision ought not to be performed until the patient has been placed in the best possible condition for operation, and that this treatment ought to be carried out throughout the healing of the wound. A much brighter aspect, however, has been thrown on these cases more recently by Abel Francon ('Lyon Méd.,' 1886, liii, 242).

He gives two cases in detail in which M. Aubert operated without any ill result, and quotes three others (Niepce, 'Lyon Méd.,' 1876, ii, 232 ; Demarquay, 'Maladies chirurg. du Pénis,' p. 245; and a personal communication from Dr. Horand). He thinks that abstention from operating ought not to be the rule, but that, on the other hand, one ought to practise circumcision in cases of glycosuria, provided that rigorous antiseptic precautions are taken. Of the cases we have collected one died of gangrene, which destroyed the skin of the genital organs, of the lower part of the abdomen, and the upper part of the thighs.

A second had severe erysipelatous swelling, followed by hæmorrhage and sloughing. The case ended ultimately in recovery.

Fournier states that a patient of his died as the result of gangrene, and that Demarquay had another ('Gaz. méd. de Paris,' 1887, t. iv, 133). There is not enough evidence to show whether previous medical treatment had any beneficial result.

This is not an operation that can always be refused. The foreskin may become so contracted at its orifice that there may be pain and difficulty in urinating, in addition to the fact that failure to keep the parts clean entails severe pruritus, with inflammation and erosion of the glans.

Although it is evident that this is a part where one cannot be certain of maintaining asepsis, Francon's cases show what can be done with careful precautions. The parts were

## *Operations for Cataract in Diabetics.*

| No. | Age and sex. | Reference. | Condition of patient. | History of the diabetes. | Medical treatment before operation or not. | Lesion. | Operation. | Complications. | Final result. | Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|
| 1 | F., 57 | Louis Thomas, 'Bull. Soc. de Chir.,' 1879, p. 176 | Thin and complete loss of strength; last stage of diabetes; profound exhaustion | History of some years; wasting; loss of strength; thirst; sugar in great abundance | Could not remain long enough to undergo preparatory treatment | Cataract | Operations for cataract | 4th day, cicatrisation complete; bandage removed. Patient began to worry very much about going home. On 8th day became unconscious suddenly | Death on 8th day; slow deep respiration; imperceptible pulse; coldness; unconsciousness | Death unconnected with operation; dependent on mental worry. Diabetic coma. |
| 2 | Child, 14 | Guersant, 'Gaz. hebd.,' 1859, t. vi, 805. Bouilly, 'Archiv. gén. de Méd.,' 1877, t. xxx, 336 | Diabetic child, developed double cataract | — | — | Double cataract | Operation | Died suddenly day of operation | Death | No lesion P.M. to account for death. Diabetic coma. Mentioned by Léoty and Bouilly. Seven other cases recovered. |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| 3 | M., 38 | Panas, 'Acad. de Méd.,' April 9th, 1889 | Bilateral cataract for two years; 49 gr. sugar in 24 hours. | — | Rigorous treatment reduced sugar from 49 to 15 gr. | — | Left eye operated on | No complications | Recovery prompt | Good results in spite of the large amount of sugar. |
| | | | Two years later returned to have other eye operated on | | Classical treatment reduced sugar from 55 to 31 gr. Antipyrin, 3 grms. a day then given; sugar disappeared | | Right eye operated on | No complications | Recovery; sugar returned as soon as antipyrin omitted | — |
| 4 | F., 63 | Panas, loc. cit. | Thin; nervous | Diabetes for 6 years; sugar, 35 gr. per 1000 | Antipyrin, 3 grms. a day, sugar fell to 3·60 gr. per litre | Double cataract | Operation | Complete success | Recovery | Good results of antipyrin treatment to be noted. |
| 5 | Young girl | Morton, 'Phil. Med. News,' 1888, vol. liii, p. 705 | — | Marked diabetes | — | Cataract | Needle operation | Sloughing and gangrene of orbital contents | Death on 3rd day from coma | — |

thoroughly washed with sublimate solution, the edges of the wound sewn with chromic catgut and dressed in one case with salicylic wool. The first case was in an advanced case of diabetes with from 3 to 10 per cent. of sugar; the wound nevertheless healed well.

The results of operations for gangrene of the extremities are somewhat complex, and we shall consider them separately later on.

*Conclusions.*—With regard to the gravity of operation, it appears—

(1) That trivial operations in youthful subjects will sometimes produce death by coma.

(2) That in older patients coma results only from severe and prolonged operations. In elderly patients with marked diabetes, the question of the necessity of a severe operation must be carefully considered. If it seems probable that the surgical lesion will carry off the patient before the diabetes will, the operation should be performed. If the diabetes be severe, the slightest operation that is possible should be performed. The operation above mentioned of excision of rectum seemed justified from the fact that no sugar had been found for two years. It would be a question in such cases of whether the comparatively simple operation of inguinal colotomy should not be performed in preference.

With regard to the site of the lesion, it appears—

(1) That in certain cases asepsis cannot be strictly maintained. Here the same questions will arise as above. Operations about the penis seem to be specially liable to gangrenous complications.

(2) Cases where asepsis is possible ought not, with our present methods, to be allowed to die of septic infection; and provided the operation be not very severe, the diabetes not in its last stages, and strictest precautions can be taken, there is every reason to expect a good result.

(3) It appears that operations about the mouth were markedly successful. Of eight operations on the lips, tongue, palate, &c., where treatment was instituted beforehand, all recovered.

*Complications occurring as the Result of Operations in Diabetics.*

Before considering the complications that may arise it will be advisable to consider, shortly, some of the changes in the tissues that predispose to these complications.

*Increased sugar in the tissues.*—Normal blood in man contains:

From . . . ·15 to ·19 parts per cent. . . . (Seegen).
„ . . . ·12 „ ·3 „ „ . . . (Frerichs).

In mild cases of diabetes, according to Seegen, the amount of sugar present does not exceed the normal, but in severe cases it may rise as high as ·4 per cent. The influence of the sugar, especially in the milder cases, in impairing the nutrition of the tissues is probably exaggerated. Nor is it by any means proved that the rapid growth of organisms is due to an increased amount of sugar in the tissues.

Bujvid ('Wien. med. Presse,' 1885), however, states that a certain amount of *Staphylococcus aureus* that will not produce any effect in a rabbit, will, if grape sugar be added to the injection, produce pyæmia and death.

*Circulatory system.*—Lancereaux and other authorities state that arterio-sclerosis is common in diabetic patients. Upon this arterio-sclerosis, says Lancereaux, depend certain pathological coincidences wrongly attributed to glycosuria—hemiplegia, dementia—common at an advanced stage of "diabète gras." To the same cause are due atrophy of the kidneys, cardiac degeneration, and dry gangrene of the extremities.

*Neuritis.*—This may be multiple or may attack particular nerves. It is the cause of loss of knee-jerk, first observed by Bouchard, 1881. Perforating ulcer sometimes occurring in diabetics is probably due to this cause. This subject is treated of towards the conclusion of the paper.

*Is healing delayed?*—We shall consider this question in—

I. *Cases that might heal by primary union,* of which we have seven cases.

| No. | Age and sex. | Reference. | Condition of patient. | History of the diabetes. | Medical treatment before operation or not. | Lesion. | Operation. | Complications. | Final result. | Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|
| 1 | M., 32 | Demarquay, 'Gaz. des Hôp.,' 1869, xlii, p. 153 | General health good; thirst, enfeeblement of energy | Diabetes detected 7 months ago; 70 grms. sugar per 1000; urine 3—6 litres in 24 hours | Régime of M. Bouchardat | Balano-posthitis; phimosis | Operation for phimosis | No hæmorrhage at time of operation; no complication; cured in 10 days | Recovery | — |
| 2 | — | 'Obs. de Pall.,' thesis of M. Brouardel, Demarquay, loc. cit. | Had preserved his stoutness | Glycosuria for long while | No mention | Phimosis | Operation for phimosis | Gangrene destroyed skin of genital organs, lower part of abdomen, and upper part of thighs | Death | — |
| 3 | M., 52 | Verneuil, Tuffier, loc. cit., obs. iv | Healthy looking; enjoying habitual good health | Strength has diminished a number of years; sugar 15 grms. per 1000 for a long while | Appropriate régime followed from time to time | Papillary hypertrophy with phimosis | Circumcision | No accidents | Recovery | The papilloma, in spite of local treatment, became epitheliomatous. |
| 4 | M., 30 | Francon, 'Lyon. méd.,' 1886, liii, p. 242 | Patient very thin; feeling of general lassitude; urine 7 litres; sugar 4·3 per cent. | Advanced diabetes; pruritus for 3 months at end of penis | No mention | Pruritus; erosions of glans; phimosis | Circumcision, with every antiseptic precaution | 24th.—Cicatrisation nearly complete; urine 5 litres; sugar 3·5 per cent. 25th.—Slight hæmorrhage from unhealed points. 29th.—Healing complete; sugar | Recovery | — |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | 49 | loc. cit. | | years; phimosis noted at same time; urine 1½ litres; sugar 5 per cent. | | and fissures for 5 years have led to extreme narrowing of preputial orifice | with most minute antiseptic precautions | immediate union; healed in 6 days | | cases great stress laid on the antiseptic precautions. |
| 6 | M., 72 | Verneuil, Tuffier, obs. iii | Vigorous man in spite of age | No symptoms given of diabetes; sugar only found some months after the patient was circumcised | Apparently diabetes was not recognised | Balano-posthitis.<br>Subsequent vegetations.<br>Subsequent epithelioma | Circumcision.<br>Cauterisation.<br>No operation on account of sugar being found | Without complications<br>Without complications | Recovery | Balano-posthitis leading to epithelioma; no accidents following circumcision. |
| 7 | M., 34 | De Beauvais, 'Gaz. des Hôp.,' 1874, 877 | Apparently stout | On account of wound going wrong, past history was inquired into. Ardent thirst; drinks enormously; polyuria. Had consulted Trousseau for obesity | No treatment immediately previous to operation | Intense balano-posthitis and phimosis; operation considered necessary | Circumcision | 3rd day.—Abundant hæmorrhage. 5th day.—Erysipelatous swelling; anti-diabetic treatment. Sloughing of preputial wound; carbuncles on thighs and buttocks | Recovery | Complications: hæmorrhage, sloughing, &c. No initial treatment. |

*Operations for Fistula in Diabetic Patients.*

| No. | Age and sex. | Reference. | Condition of patient. | History of the diabetes. | Medical treatment before operation or not. | Lesion. | Operation. | Complications. | Final result. | Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|
| 1 | M., 45 | Verneuil, 'Bull. Soc. de Chir.,' 1878, p. 479 | General condition appeared good, in spite of some attacks of gout. Big and stout patient | Sugar found on admission 26·50 gr. per 1000 | Anti-diabetic treatment for 8 days; sugar fell to 7·32 per 1000 | Anal fistula of 4 years' history | Operation with thermo-cautery | Sugar persisted 6 per 1000 in spite of treatment; slight hæmorrhage for 2 days | Wound not healed when patient was sent to Vichy | Appearance of wound improved when sugar diminished; unfavorable when sugar increased. |
| 2 | — | Landau, 'Berlin Wochensch.,' No. 43, p. 863 | — | — | Rigid diet, and sugar reduced to a minimum | Fistula for 1½ years | Operation | — | Recovery | Not many details given. |
| 3 | — | Despies | Stout patient | — | — | Fistula | Operation | — | Recovery | No details; ? diabetes. |

(1) Parotid tumour; long operation; erysipelas; hæmorrhage and death.

(2) Amputation of breast; patient up on the eleventh day.

(6) Amputation of breast; greater part of wound healed by first intention.

(7) Epithelioma of cheek and submaxillary gland; both wounds healed by first intention.

(13) Amputation of breast; healed by primary union; subsequently became septic through drainage-tube opening.

(15) Sebaceous cyst of cheek; wound suppurated; healing delayed fifteen days.

(16) Epithelioma of lower lip; union delayed for a month.

We thus see that four out of these seven wounds united by first intention. There was no delay.

Two others that ought to have been kept aseptic were complicated, in one case (1) with erysipelas, in the other (15) with some pyogenic germ.

In (16) it was impossible to keep the wound perfectly aseptic, so there is some excuse for its breaking down.

It appears that clean-cut wounds, if kept aseptic, heal without delay.

In amputations for gangrene, as will be seen later on, healing has been delayed in some cases by the condition of the vessels, septic influence being entirely absent.

II. *Cases where union by first intention is impossible.*— It is not so easy to say in these cases whether there was delay in the healing of the wound. Where the wounds were uncomplicated by septic trouble there does not seem to have been delay in healing. The only case where healing did not occur was in a fistula of four years' standing operated upon by Verneuil ('Bull. Soc. de Chir.,' 1878, p. 479). In this case Verneuil points out that whenever the sugar decreased, the appearance of the wound improved, and vice versâ. The patient was sent to Vichy with the wound unhealed.

*Cataract* operation-wounds seem to heal well, even in advanced cases. Thomas has described a case in a woman in the last stages of diabetes, where the wound healed, and the bandage was omitted by the fourth day. The woman died

from coma on the eighth day through worry about her husband.

Although the evidence here does not point to any great delay in healing in the majority of cases, we shall see, later, that diabetes markedly affects the repair of fractures.

*Hæmorrhage.*—This is a complication that the French lay considerable stress upon as occurring both during and after operations. To obviate its occurrence during operation they use the thermo-cautery, which seems in some cases at least to have been partially responsible for the secondary hæmorrhage that occurred later.

(1) Hæmorrhage on the sixth, seventh, and eighth days, set up by erysipelas.

(2) Removal of axillary glands; hæmorrhage an hour after operation, and again on the seventh day.

(10) Tongue removed with the thermo-cautery; considerable hæmorrhage on the sixth day (that is about the time of separation of sloughs).

(17) Small parotid tumour; hæmorrhage "en nappe" two hours after operation; small in amount but persistent, lasting all day.

Out of eighteen operations for growths there were four cases of hæmorrhage. Of these it is doubtful how far (1) and (10) can be considered as due to the diabetes. Hoffa specially notes in his case of excision of rectum that there was no excessive bleeding during the operation.

When it occurs in diabetic patients it is probably due to the diseased condition of the vessels, which in the later stages of the disease predisposes the individual to hæmorrhage, especially from the cerebral vessels (Lancereaux).

Amongst the other operation cases hæmorrhage is noted twice. Once in a case of circumcision for diabetic balanoposthitis, where erysipelatous inflammation set in; and in Verneuil's case of fistula, where it lasted, in slight amount, for two days. Both the cases, like (1) and (10) given above, were septic.

In wounds kept aseptic there does not seem to be a very marked liability to hæmorrhage. It seems to be more readily set up as the result of septic infection.

*Wound infection as a complication.*—There is no doubt that diabetic patients are liable on even the slightest injury to severe septic inflammation. But there is no reason why in operation cases, if proper precautions be taken, the wound should be allowed to become infected. In certain cases it is difficult to ensure strict asepsis. This is especially the case in operations on the penis, circumcision for balanitis, and amputation for epithelioma. We have two cases where gangrene followed circumcision, and many more are mentioned in surgical literature ; and in (14) amputation of the penis was followed by coma, and towards death (on the 10th day) by sloughing of the wound, and gangrene of one foot. On the other hand, the number of cases where, in wounds that ought to have been kept aseptic, infection occurred, is large in proportion. Thus (1) an operation for parotid growth was complicated by erysipelas, and death resulted. (13) shows how lack of precautions too early after operation may lead to bad results. Rosenberger operated upon a woman of sixty-one, who had been diabetic, for scirrhus of the breast. The strictest precautions were taken. On the first dressing, four days later, the wound was found to be united ; the drainage-tube and stitches were removed, and boracic acid ointment applied. Patient was considered well. Six days later patient was found to have a hot skin and a dry tongue. Rosenberger was called in, and found the lower end of wound, where drainage-tube had been, unhealed. Septic infection took place through this opening, and death occurred.

(15) Inflammation and suppuration followed removal of a sebaceous cyst of cheek. (7) A small collection of pus formed in the wound ; this healed as soon as it was let out.

Out of seven cases where wounds ought to have been kept aseptic four became infected.

There is no doubt that this increased liability to septic infection is the great danger in cases of operation in diabetics. However, in cases where strict asepsis can be maintained, wound-infection ought not to take place. We are taking it for granted that these inflammatory troubles are due to the conveyance of micro-organisms from without ; for there is no evidence that diabetes *per se* will lead to suppuration, nor is it likely that the necessary germ reaches the wound

through the blood-current, in the majority of cases at all events. A large number of cases remain where asepsis is impossible. The majority even of these seem to do well. The worst results seem to follow operations on the penis. Operations on the mouth have on the whole done well. In one case fœtid expectoration occurred after removal of a tongue by the thermo-cautery, and was thought to be due to fœtid bronchitis; and the chief danger in this class of cases is of a similar nature.

*Increase of Sugar in the Urine as the Result of Operation, or Return when previously Absent.*

This return or increase in amount of sugar is quite common as the result of operation. Out of sixteen operations, the amount was increased in one case, and recurred in six cases where, before operation, there was no sugar. In one case (10) polyuria returned after the operations but no sugar was found. The sugar in most cases is found the day after the operation, but in one case it did not make its appearance for fifteen days.

Redard ('Rev. de Chirurg.,' 1886, vi, 503) published one of these cases (8) to show the aggravating action of trauma on previous pathological states. In his case the sugar increased for six days from 10 to 25·2 grammes per 1000, then diminished again by the fifteenth day to 12 grammes. Beyond the urine, the operation seemed to have had no effect upon the system, the temperature, pulse, and other functions remaining unchanged.

It is interesting to compare this return of the sugar in diabetic patients after operation with those cases we have already described, where sugar appears, for the first time, in a person as the result of an operation.

*Coma.*—This seems to be a complication that one cannot foresee in any case. Of cases uncomplicated by a previous septic condition we have two examples (11 and 12). In both it is noticeable that there was no sugar at the time of operation. In one there had been none for two years. Both were, however, cases of severe and prolonged opera-

tion—Richelot's operation for cancer of the cervix uteri and Kraske's method of excision of rectum.

These were in patients of forty-seven and sixty years of age respectively. Of coma occurring in elderly persons as the result of a trivial operation we have no example in non-septic cases. It seems to be a complication more likely to occur in the young from such a cause. Guersant ('Gaz. hebd.,' 1859, vi, 805) gives the case of a child of fourteen who was operated upon for cataract. Death occurred suddenly on the day of operation, with no lesion, post mortem, to account for it.

Coma is mentioned more frequently as the cause of death in septic cases, carbuncle, gangrene, &c.; but how far the diabetes is responsible, and how far the septic intoxication, it is often difficult to say.

### *Influence of Diabetes on the Healing of Fractures.*

We have seen that in most cases when asepsis is maintained wounds heal readily. A certain number of cases of fracture have been noted where union was very much delayed, or did not occur at all. What proportion these form of the whole number of fractures in diabetes we cannot say, but they show the influence that diabetes may have on the process of union. It will be seen that three cases took four to five months to heal, whilst two others remained unhealed. In one of these cases a useful arm apparently resulted, with a false joint and a large amount of provisional callus.

| No. | Age and sex. | Reference. | History of the diabetes. | Fracture. | Result. | Remarks. |
|---|---|---|---|---|---|---|
| 1 | M., 35 | Verneuil, 'Bull. de l'Acad. de Méd.,' 1883, t. xii, p. 934, obs. i | Diabetes for at least 4 years | Jan. 13th.—Compound fracture of left femur. Urine, 2—3 litres in 24 hours; sugar in considerable amount | Jan. 24th.—Large superficial eschar.<br>Feb. 15th.—Ulcerated surface, healing in front, extending behind.<br>March 10th.—Much sugar, no cicatrisation.<br>April 7th.—Fragments still very movable.<br>28th.—Ulceration cicatrised.<br>March 17th.—Consolidation of bones nearly complete | Consolidation nearly complete in four months. |
| 2 | M., 57 | Ditto, obs. ii | Diabetic symptoms for 2 years; wasting; general condition bad; urine about 10 litres; large amount of sugar | Fractured neck of humerus. Entry, Aug. 4th.—Patient troublesome, loosens bandage; refuses anti-diabetic treatment | Sept. 4th.—Went out. Callus large in amount; arm seems solid and does work required well.<br>Nov. 9th.—Run over; gangrene of abdominal wall; broncho-pneumonia; death | Only provisional periosteal callus found; a marked pseud-arthrosis at site of fracture. |
| 3 | M., 54 | Ditto, obs. iii | Four years ago wasting, loss of strength; sugar found; has had gout | Fractured both bones of forearm about the middle, Sept. 14th, 1882 | Nov. 13th.—No signs of consolidation; urine contains 7·96 p.c. sugar.<br>By end of March, 1883, sugar had disappeared under treatment | Consolidation entirely wanting 9 months after fracture. |
| 4 | M., ? 55 ? 75 | Berger, 'Bull. Soc. de Chir.,' 1883, p. 309. Verneuil, loc. cit., obs. v | Diabetes for a long while; carbuncle; gangrenous ulceration of leg; recently wasting; sugar, 7·5 to 4·8 p.c. | Fractured his leg in twisting his foot in his room; oblique fracture in lower one third, March 22nd, 1883 | Sugar diminished under treatment from 4·8 to 1·7 p.c. Hæmoptysis as a complication | Union complete on 110th day. |
| 5 | — | Léoty, quoted by Bouilly, 'Archiv. gén. de méd.,' 1877, t. xxx, p. 336 | Diabetes | Broken leg | — | Healed in 5 months. |

## Part IV.

### *Gangrenous Conditions in Diabetic Subjects which affect the Surgeon.*

The tissues in diabetes are considered to have a feeble power of resistance, and to tend to necrosis, as König[1] remarks in his critique upon Roser's[2] paper. Local death in sufferers from diabetes may occur in any part of the body in consequence of a wound of the superficial tissues, the form being an acute, spreading, septic inflammation. In recent years all authors, with the exception of Roser, consider that these inflammations are due to the introduction of microbes; Roser, standing alone, avers that "diabetes forms a gap in our sepsis theory," apparently because he was not able to put a stop to local inflammations by the local applications of germicidal materials. König (v. supra) sums up the matter by saying that in a diabetic patient a gangrenous inflammation is evoked, when in a healthy person the condition would be so slight as hardly to be worthy of the term phlegmon. After wounds it does not necessarily follow that spreading inflammatory processes will ensue even in a diabetic subject; a local abscess or a limited lymphangeitis with suppurative lymphadenitis may result—as in Spencer's case of bubo following ulcerations on the penis.

Some cases of suppuration and inflammation have followed bruising or injury; again, such conditions may arise without any apparent cause.

Besides these "accidents of diabetes" there are a number of cases of spontaneous or traumatic lesions about the feet and toes which lead to the mortification of one or more toes. As they stand somewhat apart we may safely consider them under a separate heading.

Another form of local death due to inflammatory pro-

[1] 'Zeits. f. Chirurg.,' 1880, S. 138.

[2] 'Deutsch. med. Woch.,' 1880, Nos. 1 and 2.

cesses is carbuncle ("anthrax" of the French authors, sometimes transcribed by the English as anthrax, properly the equivalent of the French "charbon"). Gangrenous ulcers and inflammations are apt to occur on the penis of the diabetic patient, and Sir James Paget[1] notes a few cases of necrosis of the jaw associated with diabetes.

In the literature of the subject we meet with several memoirs which, under the title of "diabetic gangrene," treat of cases of gangrene of the toes or feet; after the above enumeration it will be seen that such a procedure is scarcely justifiable.

There is yet another condition of local death met with in diabetes, namely, "*mal perforant* or perforating ulcer," perhaps not so definitely bound up with septic infective processes.

Gangrene of internal organs does not concern us here.

(*a*) *Carbuncle.*

(*b*) *Gangrenous and inflammatory conditions of wounds generally.*

(*c*) *Gangrenous affections of feet and toes, with* mal perforant.

*Operations for carbuncle in diabetes.*—It is difficult to say how far the carbuncle and how far the operation itself is responsible for death.

Undoubtedly many cases owe their death solely to the carbuncle, this determining death sometimes from septic poisoning, sometimes by aggravating the diabetes and producing death by coma. In other cases it appears that the operation of laying open the carbuncle has determined the fatal event.

*Inflammations in diabetes in other situations than the feet.*

Since a considerable series of cases of inflammatory conditions of the feet have been published we have separated them from the present consideration.

The occurrence and association of inflammations and diabetes was first fully recognised in the fifth decade of the century, the names of Marchal de Calvi, Wagner, &c., being

[1] 'Studies from Old Case-books.'

well known in connection with it. The following two cases, being of historic interest, may be briefly abstracted:

1. Hugh S—, æt. 60, under the care of Andrew Duncan, jun.,[1] M.D, had had diabetes for more than a year.

On January 8th, 1820, he was bled from the right arm, upon which there was a boil near the elbow; this had been discharging since the 6th of January.

On the 12th he complained of pain in the arm.

13th.—Pain deprived him of sleep, the arm being much swollen and inflamed; poultices applied, and a dose of "salts" ordered.

14th.—Very little sleep, arm much swollen above and below elbow, diabetic symptoms continued; leeches and emollient cataplasms.

15th.—Some relief, but the inflammation was spreading to elbow, fever not high, 99°; pulse 104; leeches and cold saturnine lotions; urine, 23 pints.

16th.—Swelling from fingers to shoulder; complains of headache and faintness. Pulse 120, strong. Tongue clean. Mag. Sulph., ℥j. Urine, 13 pints.

17th.—Twelve leeches over pectoral muscle, fomentations of poppy heads. Bulla near elbow and general vesication. Pulse 100, small. Urine, 13 pints. Enema given. Expired tranquilly soon after visit.

*Dissection.*—No inflammation of vein, no thickening of its coats, and no effusion into it of coagulable lymph or pus. Cellular structure of arm universally gangrenous, distended with sanious fluid, and muscular fibres dark-coloured, and literally rotten. General want of fat, as is usual in diabetic cases.

2. Ann R—, æt. 23. Long suffered from scrofulous abscesses. She was bled from the right arm on May 24th, 1821, and the serum, as is usual in diabetic cases, was milky. The arm became inflamed from the wound. Three days after she was bled from the other arm; otherwise the treatment was much as in the former case. The inflammation spread, and she died on the 31st.

[1] 'Edin. Med.-Chir. Soc. Trans.,' 1824.

At the "dissection" extensive local gangrenous inflammation was found. The kidneys were thought to be rather large.

Whilst on the subject of predisposing causes Dr. Duncan remarks, "As a matter of fact connected with this subject, it is worthy of notice that the first two patients whom I lost by mortified arm (Snell and Ralston) were both affected with diabetes mellitus, and both of what is called a scrofulous constitution" (p. 590).

In both of these patients the inflammation started in wounds, and this is the usual exciting cause in other cases. In some, however, the inflammations arise from some unknown cause.

Again, there are other instances where suppuration follows woundless contusions or fractures. Kraske[1] points out that these as a rule do not suppurate. Exceptions, however, occur—as for instance, Jullien's[2] case, a man of sixty, who had a patch of gangrene on the skin of the shin from contusion, and this became inflamed. Patient said he had suffered from "diabetic eye trouble," for four years, and at the time of injury he was glycosuric; it is curious that a wound at the knee inflicted at the same time did not inflame, and the patient made a good recovery.

Verneuil,[3] who lays great stress on alcoholism with diabetes, also relates a case of a wine merchant, fifty-two years old, who had a hæmatoma of the leg without any wound. For some time he had suffered from thirst, which he endeavoured to quench by means of spirits, thanks to his occupation. The injured region inflamed, and a slough appeared; the urine contained much sugar. Within three weeks of admission he became comatose and died. At the autopsy there was some basal pneumonia, with scattered tubercles, serous effusion in the right pleural cavity, large congested kidneys, cirrhotic liver, and thickened meninges.

Berger[4] also records a case of sloughing following a sub-

[1] 'Centr. f. Chirurg.,' 1881, S. 545.
[2] 'Gaz. hebdomadaire,' 1882, p. 226.
[3] Ibid., 1877, p. 664.
[4] 'Bull. Soc. Chir.,' 1883, p. 369.

cutaneous injury in a diabetic patient. The diabetes was discovered three years before in consequence of inflammation following the cutting of a corn. The accident consisted in a severe contusion of the foot between a carriage-wheel and the kerb. Sloughing took place, but complete healing was effected in six months' time. This case is referred to among the cases of *mal perforant*, a trouble which supervened.

In the same contribution as the above-mentioned case, Verneuil gives an account of a fatal prick of the finger in an alcoholic, diabetic patient, and gives his opinion that in such cases the prognosis of inflamed conditions is extremely bad. This reminds us somewhat of one of Sir G. Humphry's many aphorisms, "Prick a brewer, and you will kill him."

We must remember, as Sonnenburg[1] remarks, that inflammations in diabetes may be of a very acute, rapid, or of a more chronic character. The following case illustrates the latter variety:

Case. *Burns; diabetes; old-standing monoplegia; procidentia uteri; suppuration of hand; amputation of forearm; discharged with sinus; death, in diabetic coma, six weeks later.*—Anne L—, æt. 58, admitted under Mr. Durham, July 9th, 1892.

*Previous history.*—Left arm suddenly paralysed in 1851. Since then the hand and arm have been useless, and the elbow has become fixed at a right angle, the fingers being rigidly bent. Patient had fair health until Whitsuntide, 1891, when she was attacked with influenza, from which she has never fairly recovered. She has wasted, and been thirsty, for about twelve months. It is stated that she drank rather freely; she looks older than her age.

On July 9th, 1892, her dress accidentally caught fire, and she was brought to Guy's Hospital.

On admission the left cheek was scorched; there was a burn destroying the skin (2 inches by 3 inches) on the inner side of the right arm; a superficial burn on the chest about two inches in diameter. There are large bullæ on the dorsal sides of the fingers, and a large raw surface (2 inches diameter) on the back of the left hand; also a small raw surface

[1] 'Berlin. klin. Wochenschrift,' 1885.

on the inner side of the left arm. Patient is very thin and wasted. Coarse, bubbly râles were heard over both bases of the lungs, the resonance not being impaired. No cardiac bruit. Slight œdema of both legs. Tongue rather red, but not the typical "raw-beef" appearance.

Urine is not retained, and dribbles constantly; this was due to procidentia uteri, which was easily kept up by a ring pessary, when the urine was controlled satisfactorily. The urine was clear, neutral, sp. gr. 1027; contained sugar, but no other abnormal constituent. On July 16th 6·25 per cent. sugar; 23rd, ·75 per cent.

The burns were cleansed and dressed daily with boric acid and vaseline, iodoform being dusted on. She was ordered the apomorphine and terebene mixture of the G. H. Pharmacopœia, 4tis horis, and one grain of codeia three times a day; anti-diabetic diet was given with ℥ij of whisky.

July 20th.—The face and hand are looking well; a slough has separated from the right arm. Patient became very drowsy, with contracted pupils and slow respiration. She does not take notice of surroundings, and scarcely talks. The urine was passed into bed; yesterday it contained 3·5 per cent. sugar. The codeia was stopped, and whisky, ℥v, ordered.

23rd.—The sugar has diminished. Patient feels much better; the burns are no longer painful. It was thought possible that the drowsiness was due to the codeia.

26th.—Not well. The left hand is very painful and swollen; it has bled once or twice enough to soak the dressings. Has been semi-comatose all day. Breath smells sweet.

29th.—All the wounds except the left hand are healing well; sloughs and pus continue to come from left hand. Her chest is clear on auscultation. The apomorphia omitted, and Liq. Strychninæ ♏v t. d. prescribed.

Mr. Durham advised patient to have the hand removed.

August 3rd.—Patient much better, to get up. Left hand still sloughing. The temperature has remained below 99°.

In the next few days she was alternately restless, rational, and drowsy, from day to day.

15th.—Sugar increasing, sp. gr. 1031.

22nd.—All the wounds nearly healed except left hand. Chest normal on auscultation.

25th.—To allay thirst a "lemonade" was prescribed:

℞ Ac. Citric. gr. xvij,
Gluside (Saccharin) gr. ½,
Aq. ad ℥j.
Sig. ℥j to be diluted with ℥j of water.

27th.—Hot boric acid fomentations to left hand:

Pil. Codeiæ gr. ½ t. d. s.

31st.—Left hand very œdematous; sanguineous pus discharged from a sinus in the palm.

September 2nd.—Patient having consented, $CHCl_3$ was administered for performing amputation. The hand was thoroughly wrapped in lint wet with 1 in 500 perchloride; the rest of the forearm being thoroughly scrubbed with perchloride lotion. Anterior and posterior flaps were raised, and the bones sawn at the insertion of the pronator teres. Hæmorrhage was arrested by torsion except the interosseous arteries, which required silk ligatures. Hot, strong carbolic lotion was used to bathe the flaps, which were then adjusted with a few interrupted sutures. No drainage-tube used. Dressed with Tr. Benzoinæ Co. and sal-alembroth wool, and splint (internal angular and a perforated-zinc cage) applied. The palm and dorsum of the hand were full of pus. The arteries were not calcareous, or noticeably thickened.

The next day the sugar was 1 per cent., on the 5th 2·5 per cent., the 6th 3·8 per cent. with a trace of albumen.

9th.—Arm is perfectly comfortable. There has been no serious pyrexia.

10th.—Stump dressed for the first time; on the radial side primary union occurred, the ulnar side was ununited and discharging pus freely. Irrigation with boric acid lotion and hot boric fomentations were ordered, the stitches being removed.

15th.—Flaps fixed with strips of strapping; no redness or œdema.

18th.—

℞ Tr. Nucis Vom. ♏x,
Ac. Phosph. dil. ♏x,
Tr. Aurant. ♏xx,
Gluside gr. ½,
Aq. ad ℥j, t. d. s.

22nd.—Stump rather better; still free discharge; from this date till October 4th, absence of sugar is noted; the discharge gradually became less, and healing and contraction progressed.

October 5th.—Reappearance of sugar. Patient feels better and stronger.

28th.—Can now walk up and down the ward without fatigue. Sugar absent from the urine for several days, then reappeared in slight amount; the patient occasionally managed to get bread surreptitiously from other patients.

November 18th.—Allowed to have a little toast. Arm dressed with dry sal-alembroth wool.

26th.—Discharged to-day at her own desire with a small sinus which discharges slightly. Patient says she feels "as strong as she ever did."

*Subsequent history.*—Through the courtesy of Dr. Elwin Harris, Medical Superintendent of St. Saviour's Union Infirmary, we are informed that patient was admitted under his care on November 29th. As she refused any treatment, she was discharged. Readmitted December 13th. Abscess opened under $CHCl_3$; evacuation of much fœtid pus; "the bones were found to be quite bare; nothing further was done, it not being considered advisible as she had a large quantity of sugar in her urine." On January 7th, 1893, she developed symptoms of "diabetic coma," and died in forty-eight hours.

*Remarks.*—In the above patient, an undoubted diabetic, it is to be noticed that the burns healed, for the most part, somewhat tardily, but without complication; the application of the weak antiseptics, boric acid, and iodoform were sufficient to prevent sepsis occurring at the granulating surfaces.

The cause of the suppuration of the hand is not clear; the nails of the tightly clenched fingers may have caused some abrasion, which allowed septic infection.

There was a marked rise in the reducing power of the urine up to the third day after the amputation, after which it fell, being almost always less than 1 per cent. It was not possible to gauge the amount of urine passed, but it would seem that the amount was not at all or but little augmented. The temperature was never raised so as to cause anxiety.

Notwithstanding the care in antiseptic proceedings, suppuration occurred; though there may have been some unseen

extension of the suppurating process, it is probably never possible in any operation, however carefully conducted, to eliminate the factor of accidental contamination. Unfortunately no experiments or observations were made to see if the same species of organism was present in the stump as in the offending hand. We have been unable to find information concerning the organisms present except in the paper by Sonnenburg,[1] where it is stated that only ordinary pyogenic cocci were present; in a paper by Schüller,[2] who found streptococci in the fatal case (Table, No. 32); and in a case observed by one of us, an alcoholic diabetic, a brewer's labourer, in which a fatal result was brought about also by streptococci, in consequence of a slight graze of the hand.

Treatment has been carried on upon ordinary lines—(1) the diabetic *régime;* (2) the evacuation of pus, the relief of tension, and the drainage and flushing of cavities. Amputation has occasionally been performed, as in the case recorded by Spencer,[3] where successful amputation of the arm was performed in consequence of the spread of a phlegmon which originated in a "spontaneous bursitis" of the olecranon bursa; in this case, as in the one we have reported, a sinus persisted for a long time.

This author also records a case of gangrenous ulceration of the penis due to diabetes, and giving rise to a bubo, which was cured after an attack of erysipelas. This is interesting from the fact that Sonnenburg and other authors assert that gangrenous processes are not known to occur before the age of *twenty-five*, and that they are rare up to *thirty-five* years. *The patient in question was only twenty-four, and a venereal origin was denied absolutely. The only other case of inflammatory conditions at an early age we have come across in our research is one recorded by Albert (Kolisko's case) of a diabetic waiter, aged twenty-three, who had an ulcerated condition anterior to the external malleolus;* it eventually responded to regulated diet, daily antiseptic baths, and dressings of powdered willow bark; the patient died about four months

[1] 'Berl. klin. Wochensch.,' 1882, S. 705.
[2] Ibid., 1888.
[3] 'Med.-Chir. Trans.,' 1892.

afterwards of general tuberculosis. The ulcer had been investigated for tubercle bacilli with a negative result. Perhaps this case should have been included amongst the foot cases; but it appears to differ from them, and is therefore mentioned here.

Gangrenous inflammatory conditions of the glans penis and prepuce are well known, and, according to Friedrich, are due to organisms which occur constantly in diabetic urine. The discovery of yeast fungi in the secretions is thought to be diagnostic of a diabetic origin. These inflammations are apt to lead to the development of phimosis (*phimosis acquisita diabetica*); for these Sonnenburg and others have performed circumcision. The results of his operations are recorded in a dissertation by Leuchert (Berlin, 1884) which we have not been able to see. This matter has been treated of above.

Acute inflammations may occur in any part of the body, but are said to be more common on the limbs than on the neck or trunk (Sonnenburg). In the former case the prognosis increases in gravity as the inflammation approaches the trunk, especially if it be a rapid form.

Amputation, especially high up in the limb, may save the patient, as in Spencer's case, but the outlook is bad. The question of amputation above inflamed tissue is dealt with more fully in the next section.

## Part V.

### c. *Gangrenous affections of the Lower Extremity, especially of the Feet and Toes.*

With the addition of four hitherto unpublished cases, we have been able to collect a total of sixty-three cases of gangrene affecting the toes and feet. In considering them and classifying them we found ourselves involved in that vicious circle dependent on the fact that glycosuria may be an antecedent (diabetes) or a secondary condition. Some of the cases are undoubtedly diabetic; of the rest, in others this is not certain, though in some few probably the glycosuria is secondary to the septic condition. The only authors who have considered this aspect in reporting actual cases of

doubtful nature are Demmler, Musset, and Max Schüller, though Israel and others have insisted upon the distinction.

Etiology.—There has been some discussion concerning the etiology of the gangrene of the toes which occurs in diabetes. Besides the diabetes itself, the following factors are or may be present:

1. *Wounds*, usually of slight or insignificant extent, becoming inflamed or causing indolent ulceration.

2. *Spontaneous inflammation* unaccompanied by any known injury. As an example we may mention the suppuration beneath hard corns, occurring sometimes in adventitious, cutaneous, bursal sacs.

3. *Arterio-sclerosis*, or other diseased conditions of the arteries whereby the blood-supply of the part is interfered with.

4. *Nerve degeneration.* This is believed to be the chief factor in the production of "mal perforant," which in some cases leads on to death of the toes.

Roser[1] believed that diabetes was the actual cause of the gangrene; but he brought forward no proof, nor has any other author, that this diathesis can cause gangrene, unaided by other factors.

In the cases before us glycosuria is combined with one or more of these four factors. Nearly all cases at one time or another become septic; infection with micro-organisms may be a prime cause in the determination of gangrene, or it may readily occur after necrosis has asserted itself, whereby many cracks are formed, and favour the entrance of microbes. Nearly all authors are agreed that the tissues are prone to inflame in diabetes, and that the altered constitution of the tissue juices makes them more favorable for the multiplication of fission-fungi. [Hunt, curiously enough, has not grasped the meaning of the word "Spaltpilz," which he translates "chink fungus."]

Arterio-sclerosis with calcareous degeneration of the coats of the arteries, in some cases of the main trunks, in others of the finer branches only or as well, is very frequently associated with gangrene of the toes. Out of the 63 cases we have collected, 24 are known to have suffered in this way; of

[1] 'Deutsch. med. Wochensch.,' 1880, Nos. 1 and 2.

the others no note is made concerning the condition of the arteries. Lancereaux considers the occurrence of arterio-sclerosis as a most potent factor. Israel and Heidenhain lay stress upon arterio-sclerosis, for if primary septic inflammation happens, it leads to a further interference with the circulation through the already hampered area, "the diminished supply of an abnormal blood" (Israel). Besides mere mechanical causes the local effect of the toxic substances engendered by the microbes, must be taken into account.

Vascular obstruction alone, of course, can and does (senile gangrene) lead to the death of a part; and the resulting condition, as has already been pointed out, readily lends itself to inoculation secondarily by microbes. Heidenhain[1] draws up four conclusions upon the relation of arterio-sclerosis and diabetes in discussing senile gangrene.

1. There is a great tendency of diabetic tissues to inflame; furthermore, they are remarkable, when inflamed, for the rapidity and facility with which they die.

2. Arterio-sclerosis and diabetes are frequently associated.

3. The frequency of gangrenous processes increases with advancing age in diabetic patients, and is not known to occur before the age of twenty-five.

4. Clinically there is a great resemblance between "diabetic gangrene" and "senile gangrene."

With regard to 1, this tendency is allowed by all authors; it is to be noticed, however, that chronic inflammations may occur as well as acutely spreading phlegmonous conditions. Albert,[2] quoting Mayer (whose paper we have not been able to obtain), remarks that organisms which are always present on the skin, &c., have a pathological influence upon diabetic tissues.

Frerichs[3] says that he has noticed sclerosis of the arterial walls, with or without valvular mischief, so frequently that he has come to the conclusion that a connection between the two is very probable. Albert relates a case of a man of fifty-two years (Table No. 22), who wounded his great toe;

[1] 'Deut. med. Wochen.,' 1891, p. 1087.

[2] 'Allgemein. Wien. med. Zeit.,' 1885.

[3] 'Ueber den Diabetes,' Berlin, 1884, p. 77.

the wound suppurated, and the toe became contracted and dead. The toe was removed with scissors. Inflammation and abscesses continued locally. In four weeks the man died. Glycosuria was noted about a fortnight after admission, and there is no evidence that the case was not one of secondary glycosuria. At the autopsy it was found that the arteries of the foot were rigid and hard, but there was no evidence that they directly caused the gangrene; and since many cases occur with similar arteries without any supervention of gangrene, Albert argues that the diabetes must have had a causal influence in determining it. He passes over the inflammatory commencement of the process entirely, and does not even show that true diabetes was present. Later in his series of contributions, he notes that senile gangrene may be dry or moist, and that in diabetic patients gangrene may be considered as senile if both the general and the arterial conditions are in keeping with this view.

Of the 63 cases before us the actual manner of commencement of the gangrene is not specified in 32. In the remaining 31 an inflammatory history is given in 30 cases (suppurating wounds, corns, &c., 17; indolent ulcers, 5; and maux perforants, 8): in the other case a history of some slight injury, from walking, is recorded. These cases give confirmation to the view that the immediate exciting cause is frequently of an inflammatory (septic) nature.

Of the cases with inflammatory beginning some are due to wounds which became inflamed, and then in direct consequence mortification in one or more toes set in; in others a callous, indolent ulcer developed at the site of the injury, which was followed later on by death of a toe. In the latter set of cases, and in the former also when the process was not acute at first, the inflammatory condition often suddenly took to rapidly spreading. Of this there are two forms, the *first* spreading along the lymph-paths (lymphangeitis), the *second* extending into the tissues without any definite association with lymph-vessels (phlegmon); both being accompanied with œdema, redness, and pain, the last often most intense.

In a few instances the trouble began without any superficial lesion, *e. g.* some inflammatory swelling, with or without

the formation of an abscess; or, again, suppuration occurred beneath the thick cuticle of a hard corn; or, lastly, death of areas of the skin occurred in patches. In some cases these healed rapidly, in others an ulcerated surface was exposed, which not infrequently became infected with virulent agents. One form of ulceration ("mal perforant") remains to be mentioned; it is of a chronic nature, though, like the other forms, it may become more actively inflamed by accidental infection. Upon this subject we shall have more to say later on.

One of the cases reported by Mendel[1] began with blisters on the feet, which arose from walking on a business tour; next day there was great pain and weakness in the legs and feet, and the plantar surface of the last phalanx (reported as "third") of both halluces, and the same phalanges of the second and third toes were black, dry, and devoid of sensation. Glycosuria (5 per cent.) was present; the urine was not markedly increased. Eventually the dead portions separated, and the ulcers healed in eight weeks. About a couple of months afterwards the patient had a second attack, from which he also recovered; it began with a pustule on the plantar surface of the hallux, and led to lymphangeitis along the thigh as well as gangrene and exposure of bones of the second toe. At the commencement of this second invasion we may note that gangrenous bullæ formed on the dorsum of one or two toes. Sir James Paget,[2] in writing of diabetic gangrene, specially mentions the occurrence of vesicles and blisters. The appearance of bullæ, filled with sanio-serous or sero-purulent fluid, are reported in several of the cases we have collected (Nos. 5, 12, 16, 20, 61). They were all septic, and probably do not differ from the bullous condition in other erysipelatoid inflammations; in one or two of the cases they are described as being as large as hen's eggs.

Age has been considered as a factor by most authors; out of the 64 cases we have collected the age is stated in 54, the maximum being seventy-two and the minimum forty.

[1] 'Berlin. klin. Wochenschrift,' 1886, p. 391.

[2] 'Studies from Old Case-books.'

| | | | | | |
|---|---|---|---|---|---|
| Between | 40 and 49 | years | there are | 13 | cases. |
| „ | 50 and 59 | „ | „ | 23 | „ |
| „ | 60 and 69 | „ | „ | 11 | „ |
| „ | 70 and 72 | „ | „ | 7 | „ |

As in many of the cases there is a history of diabetes extending over many years, it is to be inferred that gangrene is often a late complication of this many-sided diathesis.

In sex there is a marked preponderance of males over females; in 62 cases collected below, the sex is male in 53, female in only 9.

Treatment.—The following methods of treatment have been practised in the various cases:

1. *Regulated diet, with or without the aid of drugs, combined with local, expectant antisepsis.*

2. *Diet (and drugs) combined with the removal of dead structures.*

3. *Diet (and drugs) combined with removal of dead structures with a minimal portion of living tissues.*

4. *Diet (and drugs) combined with amputation some distance above the lesion of the foot.*

It will be advisable to consider the comparative effects of these means of treatment as shown by the collected cases. It may first be mentioned that probably the diet was modified in almost all the cases in the tabulated account; it is only marked *where it is specifically stated that it was so.*

The following drugs have been used:—Opium, codeia, and morphine; Carlsbad salts, Mühlbrunnen water—great favourites with the Germans; Vichy water; carbonate of soda; carbolic acid (first used by Fischer)—No. 35 is interesting, as the sugar excreted was vicarious with the symptoms of carbolic acid poisoning—arseniate of soda; antipyrin. It is not possible to compare the effects and relative merits of these drugs; the enumeration may be of interest.

### 1. *Regulated Diet and Local Antisepsis.*

There are 21 cases where this was carried out. Amongst these 15 give a diabetic history; in 6 glycosuria was discovered at the time that advice was sought.

Recovery in 9 (Nos. 4, 6, 7, 9, 12, 13, 17, 18, 21).[1]
Improvement or relief, or result unstated, in 5 (Nos. 3, 5, 10, 16, 19).
Death in 7 (Nos. 1, 2, 8, 11, 14, 15, 20).
Of the 6 without a diabetic history (Nos. 5, 7, 8, 14, 17, and 18), there were 3 recoveries, 2 deaths, and 1 unknown.

*The deaths were all associated with a spread of the local septic disease.*

Many more fatal cases might have been tabulated, but all that need be noticed is that some cases get well without serious surgical interference. In Case No. 20 amputation through the thigh was strongly advised, but not performed.

The following is a record of a case which improved under the above measures.

Case A. *Suppurating corns; glycosuria (? diabetes); gangrene of little toe; relieved.*—H. T—, M. æt. 72, admitted under Mr. Clement Lucas, 22nd February, 1890.

*Previous history.*—"Good."

*Present illness.*—For many years had a corn on outer side of the little toe of left foot; it became painful and commenced to discharge, the foot and leg becoming swollen and painful.

*On admission.*—The left foot and leg are much swollen to a short distance above the knee. Skin of the dorsum of the foot is tender and red; the little toe is swollen, and the skin on the plantar and internal aspects is white, while on the outer side is a sloughing sore, the edges of which are irregular, and in places black; the toe itself is warm and not anæsthetic. At the proximal end of the first phalanx of the hallux is a small circular sore with a thickened zone of white cuticle surrounding it. Anæsthesia is present along the line of the metatarso-phalangeal joints, on the dorsum, and also partial anæsthesia along the outer side of the foot. There is tenderness of the calf muscles when these are pinched. Both knee-jerks are present, but they are slight.

Arcus senilis in both eyes, the right eye cataractous. The pupils react to accommodation, but not to light. Pulse 84, compressible. Temperature never rose above 99°.

*Urine.*—Acid, no albumen; contains a considerable

[1] These numbers refer to Table on p. 418 *et seq.*

quantity of sugar (in amount it lay between 40 and 62 oz.; the sugar varied from 9·5 to 0 grains per ounce during his stay in hospital).

24th.—Sores are dusted with iodoform and tannic acid; the whole foot is wrapped in cotton wool.

27th.—Skin of the little toe is black and sloughy. Ordered Pil. Codeinæ gr. ¼ quartis horis.

March 4th.—Sugar disappeared from urine.

6th.—Demarcation is commencing over the second phalanx of the little toe; its extremity being black, and becoming loosened. The ulcer on the hallux is scabbed over, and all inflammatory symptoms have quite subsided. Sensation is unimpaired except on the fourth and fifth toes.

7th.—Codeine omitted.

12th.—Sugar reappeared in urine. Codeine repeated.

20th.—Urine, sp. gr. 1010. No sugar or albumen.

31st.—The dead portion of little toe separated. An ulcer formed over the fourth metatarsal bone.

April 2nd.—The last-named ulcer has healed. There is still anæsthesia on the fourth toe; otherwise sensation is normal. The fifth toe is granulating.

5th.—Went out to-day, supplied with dressings. The condition of the urine is not noted; no information is given of the condition of the arteries of the lower limb; it is to be inferred that the radial artery was not atheromatous.

There is no mention of any history of diabetes, but the continuance of the glycosuria, though intermittent, after the subsidence of the inflammation, suggests that the patient was diabetic rather than that he was suffering from septic (secondary) glycosuria.

### 2. *Modified Diet and Removal of Mortified Parts.*

There are six cases in which no further operation was performed, and of these two died with spread of the inflammation, one of them in coma. In 7 other cases (Nos. 33, 44, 45, 49, 51, 57, and 61) this measure was followed by spread of inflammation, and amputation was performed higher up the limb. The general experience of surgeons

is against removing dead parts, as it seems to kindle a slow match, which may cause a serious phlegmonous explosion. Heidenhain gives special warning against such procedure, however careful the antiseptic precautions may be; and Sir James Paget[1] says, "The less of local interference, in the way of picking off bits of slough and the like, the better."

3. *Diet and Removal of Dead Structures with a Minimal Portion of Living Tissues.*

This, like the last method, may be followed by an acute outburst of septic inflammation; though, if it does not, the procedure may not mend matters, for the wound may take on an indolent, callous condition. The following case is an example of the latter result.

Case B.—*Gangrene of the right hallux and second toe of left foot; glycosuria (? diabetes); amputation of hallux; patient went out unhealed.*—Sarah S—, F., æt. 71, admitted under Mr. Golding-Bird, March 3rd, 1890.

*History.*—Two years ago noticed pain in the right hallux after walking; on removing her boot patient noticed the toe was very red, and felt throbbing in it. Patient does not eat much, but states that she is always thirsty.

*On admission.*—She is fairly well nourished for her age. The ungual phalanx of the right hallux is gangrenous. The urine contains sugar, but no albumen or blood. Ordered Pil. Opii, gr. ss, sextis horis.

March 9th.—Mr. Lane removed the toe. Pil. Opii, gr. j, sextis horis. The wound did not show much inclination to heal; on the 12th there was a dark patch on one of the flaps.

18th.—The last stitch removed, the flaps gape dorsally.

22nd.—Black gangrenous patch on heel.

24th.—Another gangrenous patch on heel.

28th.—The wound is sloughing, the flaps being separated. The patches on the heel do not spread.

April 4th.—Sugar absent from urine.

10th.—Wound still unhealed; probing reveals bare bone.

24th.—Temperature normal. Gangrenous spots on heel

[1] Op. cit., p. 121.

nearly well. There is great pain in the foot, and the second toe of left foot is becoming gangrenous.

25th.—Continual shooting pain prevents sleep.

30th.—Wound shows no sign of healing. The second toe (left) is quite black.

May 28th.—Patient went out, condition of foot unchanged.

Altogether we have 7 such cases ; in 3 of these the result was eventually satisfactory (30, 31, and 33), in 1 the result is not known, and in 1 other a higher amputation was performed with success. The remaining 2 died with spread of the septic condition (28 and 32). All except three gave a history of long-standing diabetes ; one had had excessive thirst for three weeks previously, and one without any history; both of these died. The third was regarded by the author, Musset, as probably secondary glycosuria ; this patient recovered.

It is to be noticed that primary union did not occur in any case. One case, that of Schuster (No. 33), deserves a little more notice. The case was one in which a punctured wound of the foot became gangrenous, and on three occasions necrotic tissue was removed with all antiseptic care. The mischief spread, and amputation was advised. Up to the moment of operation the performance of Chopart's amputation was contemplated. It was thought, however, that there would be less risk if removal at a higher level were performed, and consequently Pirogoff's amputation was eventually chosen. However, the flaps sloughed, and though union took place, a fistula remained, from which bony sequestra were discharged, and complete healing did not occur until twenty months had elapsed. This cannot be regarded as a very satisfactory result.

Heidenhain, quoting from Laschke ('Dissertation,' Greifswald, 1890, not accessible to us), gives four more successful cases, one of Chopart's amputation and three of amputation of the toes. The addition of these to the above numbers gives ten cases in which the result is known to have been satisfactory in seven ; in one of these the patient was probably suffering from senile gangrene with consequent septic glycosuria.

4. *Diet and amputation some distance above the lesion.*—Following Heidenhain we may consider these in two groups, viz. cases of amputation below the knee, and cases of amputation at or above the knee. Of the former we have to add the following hitherto unpublished case (No. 37).

Case C.—*Diabetes mellitus; arterio-sclerosis; perforating ulcer of right foot; gangrene of right hallux; amputation at seat of election in leg; coma; death.*—M. H—, M. æt. 57. Illness commenced fifteen years ago, after a great disappointment and trouble in financial affairs. The first symptom noticed was craving for drink and passage of enormous quantities of urine. Was dieted for diabetes by Dr. Pavy. Two years ago cataract developed in left eye; five months ago feet became painful, and he suffered from cramps in both legs, which became cold and numb; soon after this an ulcer appeared on the left heel. Four weeks before admission to Guy's Hospital he noticed an ulcer between the fourth and fifth toes of the right foot; it caused excessive shooting pains. Latterly many of his teeth have fallen out. Admitted to Guy's Hospital under Dr. Pye-Smith on 22nd July, 1891, and transferred to Mr. Golding-Bird's care.

*Condition on admission.*—Both lower limbs wasted and feel cold. On the right foot there is a perforating ulcer between the fourth and fifth toes; the ulcer is deep, coated with black slough, and is surrounded by a wide area of inflammation extending on the dorsum of the foot. Sensation is impaired on the plantar surface of the right foot, the plantar reflex being absent on this side. Both knee-jerks absent. Urine pale, acid reaction, sp.gr. 1028. Contains both sugar and albumen. On the 8th October the side and plantar surface of the right hallux were quite black. There was no sugar in the urine, and only a trace of albumen.

October 30th, 1891.—The whole of the hallux was gangrenous, as also the skin and subcutaneous tissues of the other toes. Amputation of the leg was performed at the seat of election by Mr. Golding-Bird. The posterior tibial artery was very atheromatous, lined but not quite occluded

by white adherent thrombus; the anterior tibial and internal plantar arteries were in a similar condition.

November 2nd, 1891.—Wound dressed, healing by first intention.

3rd.—Slight sanious discharge, wound not looking so healthy. The outer flap presents a rosy flush at its upper part.

4th.—The colour has turned bluish, and the mischief is extending upon the outer flap.

8th.—The patient became comatose and died; that is, on the ninth day after operation.

Altogether in our table we have twelve cases in which amputation has been performed in the leg; five of these recovered; one was successfully re-amputated through the thigh (No. 52); the result of one is not stated, and in the remaining five death occurred. In all the fatal cases the flaps were in a septic condition; one, that of Bruch and Dentu (No. 35), symptoms of carbolic acid poisoning set in two days after the operation. The amount of sugar excretion fell from 3·62 per cent. to 1·1 per cent.; the following day the urine was again pale, and the sugar increased to 3·8 per cent. Prostration and stupor were then noted. On the fifth day the urine again became dark, with a percentage of 1·25 of sugar; the patient died in the evening.

Out of the eleven cases in which no higher amputation was performed, a long-standing diabetic history is given in five (Nos. 34, 35, 37, 42, and 43); one of these had a protracted recovery (No. 42), another had carboluria and has been mentioned above (No. 35), in a third the result is not given; the other two died. Among the remaining six cases, two (Nos. 36 and 41) were considered by the authors, Demmler and Musset, as secondary septic glycosuria. They both recovered; Demmler's case is more fully given below. Two more (Nos. 40 and 41) are probably to be placed in the same category, for the glycosuria ceased after amputation; they both made a good aseptic recovery. The two remaining (Nos. 38 and 44) cannot be assigned to either heading; they both ended fatally. Alcoholism is recorded in three cases (Nos. 40, 42, and 44); only one (No. 44) succumbed. Heidenhain quotes one successful case from Laschke ('Dissert.,' Greifs-

wald, 1890). By far the most interesting of these cases is that of M. Demmler, a man of 51, who took alcohol, but not sufficient to be termed alcoholic, and who had had a severe attack of intermittent fever in the summer of 1882. On November 22nd, 1883, he was exposed to cold and wet, following which mortification of both feet set in, accompanied with severe pain. On December 20th the urine was found to contain much sugar; this was known to have been absent four months previously, and Demmler considered that the glycosuria was dependent upon the gangrene of the feet. Anti-diabetic treatment was instituted, and arseniate of soda given. On January 5th, 1884, on the supposition of secondary glycosuria the left leg was amputated at the seat of election through "altered tissues;" hæmorrhage occurred, but was controlled by means of perchloride of iron. The sugar fell from 1·1 per cent. to ·7 per cent. on the next day, when gangrene of one flap had already asserted itself. Owing to the improved condition of the patient the right leg was amputated by the circular method on January 23rd, as in the left leg, which was now trimmed up, nearly three centimetres of the tibia and fibula being removed. In three days the sugar dropped to ·4 per cent., and in ten days had completely disappeared; moreover it did not reappear with abundant nourishment. The stumps were completely healed by the end of March. Demmler remarks that the judicious analysis of the cause of glycosuria was probably the means of saving the patient's life, and insists on the importance of determining *whether glycosuria is dependent upon a diabetic diathesis or upon suppurative foci as an epiphenomenon.* The other case in which the cause of the glycosuria was discussed by the surgeon (No. 41) subsequently had gangrenous toes removed with success from the other foot (No. 30). Heidenhain records the case in which re-amputation was necessary in the thigh; the glycosuria having disappeared by treatment, amputation of the leg was performed, almost complete gangrene of the flaps occurred without the onset of signs of sepsis; re-amputation above the condyles was successful.

*Amputations through the thigh.*—Of these a considerable number have been reported, the majority having been con-

tributed by Heidenhain. We have only one case to add which is as follows.

Case D. *Gangrene of left foot; extensive arterio-sclerosis; glycosuria; amputation through thigh; death third day.*—S. T—, male, æt. 63, admitted under Mr. Davies-Colley on May 12th, 1890.

*History.*—At the end of December, 1889, the left foot swelled, especially the toes and inner part of foot. Was told he had a soft corn, which was touched with caustic; a fortnight later he consulted a herbalist, during whose treatment a great part of the swelling disappeared. The skin broke early in the course of the inflammation, and he noticed an offensive odour.

On March 6th, 1890, patient went to Camberwell Infirmary, whence he was transferred to Guy's on May 12th.

The pain, described as agonising, of the foot has prevented sleep for the last two months. He has suffered much from thirst, but not from abnormal appetite.

*On admission.*—Skin of left foot abnormally white, and on its anterior and outer parts the four outer metatarsal bones are exposed to their bases. The abductor hallucis is exposed for its anterior two thirds; the metatarsal bones are also exposed in the sole; the hallux is shrivelled and dry; there is a good deal of discharge from the under surface of the foot, accompanied with much fœtor; in a word, a condition of moist gangrene.

The urine was acid; sp. gr. 1032; sugar present; albumen absent.

13th.—Liq. Morph. Hydrochlor. ℥x, nocte maneque.

15th.—Hæmorrhage occurred; it was controlled by means of artery forceps and tannic acid.

16th.—Sugar present 1·06 per cent.; chloroform administered. Amputation was performed at the junction of the lower and middle thirds of the thigh. Very little bleeding occurred. Towards the end of the operation the pulse became slow and the breathing bad. Examination of the limb showed that the popliteal, posterior tibial, and peroneal arteries were very much diseased, being reduced almost to calcareous tubes, the calibre of which was extremely small. The hæ-

morrhage which occurred on the 15th inst. appeared to have come from the internal plantar artery. No thrombosis discovered.

17th.—Very feeble; pulse 120, small, and thready; resp. 26; temp. 100·4°; lies in dazed condition, with eyes half closed; at intervals he calls out with rambling remarks.

18th.—Patient has not rallied; condition not markedly changed; pulse 130, resp. 30; at midday, temp. rose to 103·6°, and death occurred at 4.30 p.m.

*Post-mortem examination by the Surgical Registrar.*—Kidneys rough surface, considerable interstitial change; liver fatty and large; pancreas small; one testicle showed fibroid change. Locally the femoral artery is noted as diseased and the flaps sloughy; the tissues easily separable from the bone.

The only evidences of diabetes recorded are (1) glycosuria, (2) thirst, and (3) a small pancreas. The direct cause of the gangrene appears to be the obstructed condition of the vessels; it seems to have commenced without any external lesion. The septic condition may have caused the glycosuria, but this does not appear probable.

Altogether we have nineteen cases of amputation at or above the knee, two through the knee (Nos. 49 and 50), one Gritti's amputation (No. 45), and the rest through or above the condyles of the femur.

Double amputations occur twice, in each case with fatal result (Nos. 47/48 and 55/56).

Re-amputations occur three times—once after Chopart's amputation (63); once after amputation of the leg, a case which has been already mentioned (52); and once after amputation through the condyles (53).

Of the nineteen cases eight died and eleven recovered. The cause of death was of an independent nature in two cases—(i) œdema of lungs (48), and (ii) cardiac failure (54). The fatal issue was probably due to shock in two cases (46 and 49); in two others the wound remained aseptic, and death occurred in coma on the fifth and sixth days respectively (56 and 58), the former of these having already undergone amputation through the other thigh. The case in which Gritti's amputation was performed died with metastatic

abscesses, and suggests the designation of pyæmia. The unnecessary exposure of cut bony surfaces—one of the bugbears of the old pre-antiseptic days—may have been the *fons et origo mali.*

Amongst these cases, seven give a definite history of diabetes (Nos. 46, 47, 48, 49, 55, 56, and 63), comprising two recoveries, one death from independent cause, and two cases in which the condition was probably almost hopeless at the time of operation.

Of the remaining twelve cases twice the sugar disappeared, and was not found again eight months (51) and three years (61) after, respectively; both of these recovered, and in both the glycosuria was probably an epiphenomenon. The rest must be considered doubtful in nature, especially one of Heidenhain's cases, which is described as a diabetic, but no sugar was ever found (54). Owing to the meagreness of our information we can make no useful comparison of the results in the two classes of cases.

The results of these two methods of treatment may be thus summarised:

| | LEG. | | THIGH. | | Independent |
|---|---|---|---|---|---|
| | Recovered. | Died. | Recovered. | Died. | deaths. |
| Diabetics | 2 | 2 | 2 | 4 | 3 |
| Secondary glycosurics | 4 | 0 | 2 | 0 | 0 |
| Glycosurics of doubtful nature | — | 2 | 6 | 3 | 1 |
| Totals | 6 | 4 | 10 | 7 | 4=31. |

These numbers are not, on the whole, extremely encouraging, unless it be remembered how grave the prognosis is in all such septic cases. Two points, however, have been proved: (i) that amputation can be performed with aseptic results and primary union; (ii) that the shock of such amputations does not necessitate the onset of coma. The majority of the above cases which died of coma were also exposed to septic absorption. It is by no means clear how far these cases owed their comatose condition to the sapræmia (sephthæmia), and how far to the onset of diabetic coma. There appears to be an irresistible fascination about recording all fatal cases in diabetes with a comatose termination as cases of diabetic coma.

One further operative procedure remains to be mentioned.

Dr. T. G. Morton[1] performed nerve section in two cases of diabetic gangrene of the toes for the relief of excruciating pain. In one case partial relief resulted after section of the posterior tibial nerve; in the other complete relief resulted from section of the sciatic nerve: the gangrene was not influenced by the operation, but slowly spread to four inches above the ankle, when the dead parts were removed without encroaching on the living tissues; improvement resulted. The patient died eight months after of dysentery. No statement is made concerning any diabetic history or symptoms beyond the presence of glycosuria.

### *Summary of Opinions on Gangrenous and Inflammatory Conditions.*

It will be well now to quote the opinions given by various surgeons; we may take it as understood that many have declined to amputate in diabetes for fear of an anticipated fatal result, especially in pre-antiseptic days. König[2] drew up the following conclusions in 1887 in writing of "Diabetic Gangrene:"

1. Diabetic patients (Zuckerkranken) have a great tendency to inflammatory and inflammatory-necrotic processes.

2. In all cases of spontaneous or traumatic lesions becoming gangrenous or phlegmonous, the urine should be tested for sugar. [We might add that steps should be also taken, in the event of sugar being discovered, to determine whether secondary glycosuria or true diabetes is present.]

3. Inflammations and necroses in diabetics must first be treated by general anti-diabetic measures. Antiseptic procedures which would be effectual in normal individuals are of no use; to be effectual they must be of the most searching description.

4. Amputations for inflamed limbs must not be undertaken so long as the local and general symptoms of diabetes continue.—This the writer modified, after having performed successful amputations (v. supra), to the following:—In cases of diabetic gangrene, if, in spite of local antisepsis, and anti-

[1] 'Philad. Med. News,' vol. liii, 1888, p. 705.
[2] 'Zeitschr. f. Chirurgie,' 1887, S. 225.

diabetic treatment, both the diabetes and the local phlegmon fail to improve, and danger is to be expected by further delay, a radical operation (*sc.* amputation) should be performed, with the *most scrupulous antiseptic* care, in order to save the life of the patient.

Heidenhain [1] comes to the following conclusions :

1. The tissue disturbance in diabetes, and possibly the increased amount of sugar in the fluids, afford a good soil for the growth of microbes ; hence arise the dangers of otherwise slight injuries, which are apt to run on to death of the tissues ; and the frequency of phlegmono-gangrenous and carbuncular processes.

2. Diabetes is combined very frequently (and often, too, at comparatively early ages) with severe arterio-sclerosis.

3. The so-called "diabetic gangrene" of the lower extremity in a large number, probably a majority, of cases is due to arterio-sclerosis, just as is the case in senile gangrene.

4. Senile gangrene is due to primary inflammatory disturbance of the local circulation, or to a thrombotic plug in the main artery.

5. When the gangrene only affects two or three toes, demarcation should be waited for and encouraged by local antiseptics, by elevation of the limb, and also by massage ; the removal of dead parts by means of scissors and forceps is frequently followed by further gangrene, even though demarcation was pronounced. When the gangrene spreads to the dorsum or the sole of the foot, amputation should be performed above the condyles, the flaps being cut as short as necessity will allow. The prognosis is far more favorable if no secondary suppuration and fever have already developed. Occasionally, however, less severe operations have been successful.

Roser,[2] who does not believe in the septic nature of the inflammations (which he regards as due to the diabetes *vi suâ*), relies solely upon dietetic treatment.

Israel[3] advises that—1. All unnecessary surgery (decorative, or for benign neoplasms) is to be avoided. 2. Most careful

[1] 'Deutsch. med. Wochenschrift,' 1891.

[2] Ibid., 1880, Nos. 1 and 2.

[3] 'Berlin klin. Wochenschr.,' 1882, S. 705.

antisepsis combined with the avoidance of injury to vessels, of the use of Esmarch's elastic bandage, of tension of stitches, and the like, is to be practised. 3. When delay is possible, await the decrease of the sugar excretion, although the tissues still remain in a perverted condition. 4. In necrotic inflammations of the lower limbs antiseptic dressings and irrigations of the wound should be employed with elevation of the limb. 5. In "diabetic gangrene," as a rule, amputation should not be practised; if the septic effect upon the patient renders amputation necessary it should be performed at the knee-joint, in order that no bone marrow spaces may be opened.

Mendel[1] draws attention to (1) the administration (*a*) of regulated diet to improve the blood; (*b*) of cardiac tonics to improve the circulation; (2) local antisepsis (especially iodoform); (3) the bad prognosis of operative interference.

Schüller[2] thinks that diabetes leads to gangrenous conditions (intermediate between *senile* and *septic progressive gangrenes*), only in so far as it evokes arterial disease even in young and apparently otherwise healthy individuals. Before operating, the danger of surgical interference should be put clearly to the patient, and general anti-diabetic treatment should be insisted upon. In phlegmonous conditions thorough local surgery and antisepsis (thorough syringing, hot baths, and hypodermic injections of mercury perchloride; powders for causing desiccation appear to be of little use) are more important than general treatment; if the inflammation stops, or demarcation occurs, removal through sound tissue should be undertaken.

Lancereaux also attaches the very greatest importance to the presence of arterio-sclerotic changes, and finds that the majority of "accidents" are traceable to this complication of diabetes.

Verneuil[3] lays great stress upon alcoholism, and suggests a separation of "alcoholic diabetics." He considers the prognosis in inflamed conditions far worse in "alcoholic diabetics" than in cases of simple diabetes or alcoholism.

[1] 'Berlin klin. Wochenschr.,' 1886, S. 391.
[2] 'Ibid., 1888, SS. 944, 964, 989.
[3] 'Gaz. hebdom.,' 1877, p. 664.

Demmler[1] insists on the importance of determining whether the glycosuria is an epiphenomenon dependent upon the existence of gangrenous foci. If it is, the removal of such foci is strongly indicated.

Godlee[2] considers that atheroma and peripheral neuritis do not contra-indicate amputation in diabetes. The arterial disease makes the prognosis less favorable, while the nerve condition has little effect.

Sonnenburg[3] insists on the importance of a correct diagnosis of diabetes, or secondary glycosuria; and emphasises the frequent resistance of diabetic inflammation to treatment that is successful in ordinary circumstances. In gangrene of the toes and foot he recommends the use of antiseptic desiccating powders, such as iodoform, zinc oxide, and naphthalin, with cotton-wool dressing. A well-regulated diet often has a marked effect.

Spencer[4] gives the following causes of failure in amputation for diabetic gangrene:

1. Occlusion of the arteries.
2. Inclusion of inflamed tissue in the flaps, or inoculation of the wound at the operation.
3. Delay until the gangrene has advanced too far, or until albuminuria has set in.

Consequently he advises that amputations be performed high up in the limb, and that delay caused by the trial of diet and drug treatment should be avoided.

### *Conclusions.*

Finally, we come to the following conclusions:

1. It is desirable to find out whether *secondary glycosuria* or *diabetes* is present, if possible.

2. The meagre state of our knowledge concerning the microbes present in the inflammatory lesions of glycosuric patients is much to be regretted, and sadly needs amending.

3. With expectant treatment, by means of local anti-

[1] 'Bull. Soc. de Chir.,' 1883, p. 506.

[2] "Report of Med.-Chir. Soc.," in 'Brit. Med. Journ.' and 'Lancet,' Oct. 29th, 1892.

[3] 'Berlin klin. Wochenschrift,' 1885, S. 520, § 542.

[4] 'Med.-Chir. Soc. Transactions,' 1892.

## *Gangrenous Affections of the Feet and Toes associated with Glycosuria.*

A.S. = Arterio-sclerosis; "D." = ? Diabetes; D. = Diabetes; θ = Polydipsia; G. = Glycosuria; P. = Polyuria; W. = Wasting.

| No. | Surgeon or reporter. | Age and sex. | Diabetic history; albumen. | Bodily condition; alcohol; arterio-sclerosis. | Condition calling for treatment. | General treatment. | Surgical treatment. | Progress. | Result. | After-history. |
|---|---|---|---|---|---|---|---|---|---|---|
| 1 | Bowlby, i | F. 58 | D. some years | — | "Mal perforant;" gangrene of foot | — | — | Spread of inflammation | Coma; death 4 days | — |
| 2 | „ iii | M. 56 | D. | — | Traumatic ulcer; "mal perforant;" foot gangrene | — | — | Spread of inflammation | Sepsis; death | — |
| 3 | „ iv | M. 44 | D. | — | "Mal-perforant;" gangrene 5th toe | — | — | Spread of inflammation | Not stated | — |
| 4 | Champouillon, ii | F. 47 | G. 18 mos. | — | Gang. patch 5th toe; inflammation | — | — | Separation of slough | Healed in 44 days | — |
| 5 | Dupuy | F. 64 | G. | Strong constitution | Œdema, pain, bulla; gangrene hallux | Diet; opium | — | Local trouble unaffected | Not stated | — |
| 6 | Favrot | M. 59 | D. 16 yrs. intermittent | Strong, healthy | Diffuse inflammation both feet; right foot suppurated | Diet, &c. | Incision | Improvement | Healed 4 months | — |
| 7 | Griesinger | — | G. θ | A. S. | Inflammation; toe gangrene | Diet | — | Healing | Recovery | — |
| 8 | Hodgkin | — | G. | A. S.? | Toe and foot gangrene, "senile-like" | Diet, &c. | — | Progress of inflammation arrested | Death in 6 or 7 months | — |
| 9 | König | F. 50 | D. | Well nourished | Toe gangrene | Diet | — | Toes cast off; healing | Recovery | — |
| 10 | Clement Lucas (Case A) | M. 72 | D. | — | Suppurating corn; toe gangrene | Diet; codeia | — | Toe cast off | Relieved | — |
| 11 | Landouzy | M. 50 | D. many years | — | Spontaneous gangrene, lower limbs | — | — | — | Death in few days | — |
| 12 | Marchal de Calvi | M. 40 | D. 10 years | — | Bulla; gangrene of toes | Diet; Sod. Bicarb. | — | Toe cast off; healing | Recovery | 2 years after neglected treatment; foot gangrene too |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| 13 | Mendel | M. 61 | D. | Fair | Blisters from walking; dry toe-gangrene, healed; recurred with lymphangeitis | Diet | Camphor dressing | Improvement | Recovery | |
| 14 | Musset | M. 68 | G., no history of D. | Corpulent | Spontaneous gangrene of foot; sphacelation of foot | — | — | Spread of gangrene | Death after 50 days | — |
| 15 | „ | M. | D. 5 or 6 years | — | Spontaneous gangrene of both feet | — | — | — | Death from gangrene | — |
| 16 | „ | M. 52 | θ and P. long time, G. | Powerful | Bulla; indolent ulcer; toe-gangrene | — | — | Spread of gangrene | Not stated | — |
| 17 | Roser | M. | G. | Powerful | Toe-gangrene | Diet | — | Rapid improvement | Recovery | — |
| 18 | „ | M. | G. | Powerful | Toe-gangrene | Diet | — | Rapid improvement | Recovery | — |
| 19 | Saundby | M. 57 | θ 18 mos., P. 1 year, albumen | — | Toe-gangrene; no knee-jerks | Diet; opium | — | Improvement | Relieved | — |
| 20 | Schuster, ii | M. 50 | Treated for D. 3 years ago | Healthy, small | Removal of corn; sinus; erysipelatous swelling; bullæ; toe-gangrene | — | Incision | Spread; amputation of thigh advised; delirium, somnolence | Sepsis; death | — |
| 21 | Spencer | F. | D. 5 years | — | Toe-gangrene threatened thrice | Anti-diabetic | — | Improvement | Recovery | — |
| 22 | Albert | M. 52 | P. and G. after admission, trace alb. | Wasting lately, A. S. | Suppurating wound of toe; toe-gangrene; septic condition | — | Removal of dead parts, incisions | Pyrexia | Sepsis; death | P.M.—Granular kidneys. |
| 23 | „ | M. 63 | G., trace alb. | Very powerful, alcohol, A. S. | Suppurating corn; toe-gangrene | — | Removal of necrosed parts | Lymphangeitis | Death in coma | — |
| 24 | Duplay | M. 47 | D. | — | "Maux perforants;" gangrene of hallux | — | Incisions, removal of sequestrum | Improvement | Recovery | — |

| No. | Surgeon or reporter. | Age and sex. | Diabetic history; albumen. | Bodily condition; alcohol; arterio-sclerosis. | Condition calling for treatment. | General treatment. | Surgical treatment. | Progress. | Result. | After-history. |
|---|---|---|---|---|---|---|---|---|---|---|
| 25 | Kirmisson | M. 58 | D. 3 years | — | "Mal perforant;" gangrene of toes | — | Disarticulation of toes | Suppuration | Not stated | — |
| 26 | Morton | F. 70 | "D." | — | Gangrene of foot; neurotomy of great sciatic nerve | — | Removal of dead tissues | Some improvement | Improved | Died of dysentery 8 months after. |
| 27 | Roser | M. 48 | D. | — | Advancing toe gangrene | Diet | Healing, removal of toes | G. reduced to trace | Recovery | Remained on diet. |
| 28 | Bowlby, ii | M. 44 | D. | — | "Mal perforant;" gangrene of toes | Diet, &c. | Syme's amputation | Sloughing of flaps | Sepsis; death | No P.M. |
| 29 | Golding-Bird and Lane | F. 71 | D. | Fairly nourished | Pain; swelling; gangrene of ungual phalanx of hallux | Diet; opium | Amputation of hallux | Non-union of wound; gangrene on 4th toe and heel | Went out unhealed | — |
| 30 | Musset | M. 59 | "2nd G." | Thin, once robust | Erysipelas of left foot; indolent ulcer; gangrene of toes | — | Amputation of first four toes at metatarso-phalangeal joint | Good | Recovery | Same case as No. 41. |
| 31 | Paget | M. 60 | D. | — | Gangrene of foot | — | Amputation of foot | Good | Recovery | Died of D. gangrene of other foot 3 years later. |
| 32 | Schüller | M. 51 | θ 3 wks., G., alb. | Thin, A. S., well nourished | Suppurating corn; toe-gangrene | Diet; antipyrin | Incision, later Pirogoff's amputation | Good at first; lymphangeitis | Sepsis; death | No P.M. |
| 33 | Schuster, i | F. 60 | G. found late | Very corpulent | Gangrenous inflammation of puncture; toe-gangrene | — | Removal of gangrenous tissue thrice, later Pirogoff's amputation | Sloughing of flaps | Healed in 20 months | Well 2 years after. |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| 34 | Bowlby, v | F. old | D. | — | Moist gangrene of foot | — | Amputation (where?) | Sloughing of flaps | Septic death | pheral neuritis. |
| 35 | Bruch and Dentu | M. 66 | D. 16 years | — | Inflamed wound, hallux; gangrene spread on foot | Diet, &c.; unsuccessful | Amputation above malleoli | Carbolic acid poisoning; diminution of G.; pyrexia | Coma; death 5th day | — |
| 36 | Demmler and Chauvel | M. 51 | "G. sec." | Malarial cachexia | Gangrene of both feet | Diet; arseniate of soda | Amputation at seat of election, both legs; re-amputation of one | G. ceased | Recovery | — |
| 37 | Golding-Bird | M. 57 | D. 15 years, alb. | A. S. | "Mal perforant;" gangrene of hallux | Diet, &c. | Amputation at seat of election | Sloughing of flaps | Coma; death 9th day | — |
| 38 | Kirmisson, ii | M. 53 | G. | A. S. | "Mal perforant;" gangrene of toes | — | Amputation lower third leg | Suppuration | Death 8th day | — |
| 39 | König, i | M. 70 | G., P. | Healthy, A. S. | Inflamed toe; toe-gangrene | Diet | Amputation at middle leg | Aseptic; G. ceased next day | Recovery | — |
| 40 | „ ii | M. 40 | θ W. 9 months, P., G. | Well nourished, alcohol, A.S. | Dry toe-gangrene, with phlegmon | Diet; Carlsbad; carbolic acid | Amputation of leg | Aseptic; G. ceased | Recovery | No G. on discharge. |
| 41 | Musset | M. 58 | "2nd G." ? | — | Toe amputated for gangrene 2 years ago; recurrence of gangrene, right foot | Apparently not dieted | Amputation below seat of election | No complication | Recovery | Same as No. 30. |
| 42 | Spencer [Langton] | M. 65 | D., alb. | Alcohol, A. S. | Toe-gangrene | — | Amputation lower third leg | Slight gangrene of flaps | Protracted recovery | G. persistent. |
| 43 | Saundby | M. 64 | D. 1 year | — | Double toe-gangrene; removal of necrosed parts 5 years; spread to ankle | — | Amputation of leg | No further history | Not stated | — |
| 44 | Willett and Walsham | M. 47 | G. | Alcohol | Spontaneous gangrene of toes | Diet | Toe removed with scissors, amputation of leg | Sloughing of flaps; spread of gangrene | Death, sepsis, 14th day | — |

| No. | Surgeon or reporter. | Age and sex. | Diabetic history; albumen. | Bodily condition; alcohol; arterio-sclerosis. | Condition calling for treatment. | General treatment. | Surgical treatment. | Progress. | Result. | After-history. |
|---|---|---|---|---|---|---|---|---|---|---|
| 45 | Albert | M. 53 | G. | A. S. | Alleged frostbite; ulcer; toe-gangrene | Diet; Carlsbad | Removal of necrosed parts; spread; Gritti's amputation | Suppuration; metastatic abscess formation | Sepsis; death | No P.M. |
| 46 | Davies-Colley | M. 63 | D., no alb. | A. S. | Inflammation and gangrene of foot | Diet; Liq. Morph. Hydrochlor. | Amputation middle and lower third thigh | Sloughing of flaps; did not rally from operation | Shock; death 3rd day | — |
| 47 | Heidenhain, vii | M. 58 | D. 5 years | A. S. | Foot-gangrene; inflamed leg | Diet | Transcondylar amputation | Healed by first intention | Recovery | Vide next. |
| 48 | ,, viii | M. 58 | D. 5 years | A. S. | Gangrene of heel | Diet | Transcondylar amputation | Wound healed; œdema of lungs | Death | No P.M. |
| 49 | ,, ix | M. 49 | D., breath sweet | A. S. | Foot-gangrene; somnolence | Diet; Carlsbad; salicylate | Removal of toes, amputation at knee | Remained comatose | Death in 24 hours | No P.M. |
| 50 | ,, x | M. 59 | G. | A. S. | Gangrene of hallux | Diet | Incision; amputation at knee | Healed in 28 days | Recovery | — |
| 51 | ,, xvi | M. 49 | G., P. | — | Gangrene of hallux; ulcer on other foot | Diet | Removal of toe; amputation, supra-condylar | Healed in 61 days | Recovery | No G. 8 months after |
| 52 | ,, xviii | M. 71 | G., trace | A. S. | Gangrene of heel | Diet | G. reduced to 0; amputation leg; re-amputation, supra-condylar | Healed in 45 days | Recovery | — |
| 53 | ,, xxiii | M. 54 | G., trace alb. | — | Pustule; toe-gangrene; rigors | Diet | Incision; amputation, transcondylar; re-amputation, supra-condylar | Sequestra removed; healed in 91 days | Recovery | — |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| 54 | „ xxiv | M. 52 | "D.," no G. recorded, much alb. | A. S. | Corn; ulcer; toe-gangrene | Diet | Amputation, supra-condylar | Healed in 14 days; 2nd gangrene | Death, cardiac, 58th day | P.M. |
| 55 | „ xxv | M. 54 | "D.," alb. | A. S. | Ulcer; toe-gangrene; pyrexia | Diet; Carlsbad | Amputation, transcondylar | Healed in 52 days | Recovery | Vide next. |
| 56 | „ xxvi | M. 54 | "D.," alb. | A. S. | Gangrene of other heel; phlegmon | Diet; Carlsbad | Incisions; supra-condylar amputation | Wound aseptic | Coma; death 5th day | — |
| 57 | „ xxvii | M. | D. for years | — | Gangrene of toes; no pyrexia | Diet; Carlsbad | Disarticulation toes; amputatio femoris | Healed by first intention | Recovery | — |
| 58 | „ xxix | M. 70 | G., alb. | — | Dry toe-gangrene; no pyrexia | Diet; massage | Amputatio femoris | Wound aseptic | Death in coma 6th day | — |
| 59 | Hunt, i | M. 70 | G. | A. S. | "Diabetic gangrene" of foot | — | Amputatio femoris | Rapid disappearance of G. at times | Recovery | — |
| 60 | „ ii | M. 40 | G. | A. S. | "Diabetic gangrene" of foot | — | Amputatio femoris | Rapid disappearance of G. at times | Recovery | — |
| 61 | Hutchinson | M. 48 | G., trace alb. once | A. S. | Bullæ; abscesses; toe-gangrene | — | Removal of phalanges; supra-condylar amputation | Healed first intention | Recovery | No G. 3 years after. |
| 62 | Sonnenburg | M. | G. | Powerful, alcohol | Inflamed wound of hallux; inflammation spread | — | Amputation thigh on 8th day | Bad | Coma; death soon after | No P.M. |
| 63 | Zeller | M. 63 | D., alb. | A. S. | Toe-gangrene; demarcation | — | Chopart's amputation; 3 months afterwards amputation lower third thigh | Healed by first intention; fistulæ; local infection; erysipelas | Recovery | Trace of G., much albumen on discharge. |

sepsis and anti-diabetic measures, some cases get well, even when there are signs of inflammation in the foot or leg; so that the surgeon should not resort to amputation at once.

4. Since the septic process may engender glycosuria, or exaggerate an already present glycosuria, the removal of the inflamed focus—*e.g.* by amputation—is likely to lead to amelioration.

5. If amputation be undertaken, it is necessary to be careful in the antiseptic routine. The fear of diabetes which originated in pre-antiseptic days may apparently influence us less in these days of antiseptic surgery.

6. The site of amputation must be judged of (i) so as to avoid including infected tissues in the stump; (ii) so as to make certain that the arterial supply to the stump may be sufficient, or more than sufficient. When the arteries are extensively diseased, it is to be expected that, in amputations of the lower extremity, the blood-supply will be more probably sufficient if the operation is performed before the main vessel has broken up into a number of large branches; hence arises, in part, the success of operations at or above the knee-joint.

7. In limbs poorly supplied with blood, and also, perhaps, the seat of nerve-deteriorations, when amputations are performed, such procedures as osteoplastic operations (Pirogoff's, Stokes-Gritti's amputations) are to be avoided, especially when sepsis is likely to ensue.

8. The flaps at the operation should be treated with extraordinary delicacy.

9. The term "gangrene" is used indiscriminately for the anæmic death of parts as well as for acute inflammatory (septic) conditions—a matter which surely needs remedy by the heads of the profession.

### *Bibliography of Toe Gangrene.*

1. Albert.—"Ueber Diabetes Mellitus und Gangrena Senilis," 'Allgem. Wiener med. Zeit.,' 1885, Hft. 1, 2, 4, 5, 8, and 9.

2. Bowlby.—'Treatise on Injuries and Diseases of Nerves,' London, 1889.

3. Bruch and Denton.—'Bulletin de la Soc. de Chirurgie,' 1885, p. 642.

4. Champonillon.—"De la Gangrène spontanée compliquée du Diabète," 'Gaz. des Hôpitaux,' 1852, p. 190.

5. Chauvel and Demmler.—"Gangrène des deux pieds," &c., 'Bull. Soc. Chir.,' 1883, p. 504.
6. Dupuy.—"Gangrène glycosurique," 'L'Union médicale,' 1861, p. 506.
7. Favrot.—"Variété rare de gangrène inflammatoire chez en sujet diabétique," 'L'Union méd.,' 1861, p. 427.
8. Frerichs.—'Ueber den Diabetes,' Berlin, 1884.
9. Godlee.—'Lancet,' 1892, ii, p. 1197.
10. Griesinger.—'Arch. f. physiol. Heilkunder,' 1859.
11. Heidenhain.—"Ueber die Behandlung der senilen Gangrän der unt. Extrem. insbes. bei Diabetikern," 'Deut. med. Wochenschr.,' 1891, SS. 1087, 1111, 1123, 1163.
12. Hunt.—"Diabetic Gangrene," 'Philad. Med. News,' 1888, ii, p. 685.
13. Hutchinson.—"High Amputation in Senile Gangrene," 'Trans. Roy. Med.-Chir. Soc.,' 1887, p. 97.
14. Israel.—"Enziehungen der Diabetes Mellitus zur Chirurgie," 'Berl. klin. Wochenschrift,' 1882, S. 705.
15. Kirmisson.—"Du Mal perforant chez les Diabétiques," 'Arch. gén. de Med.,' 1885, xv, p. 44.
16. König.—Report of Roser's paper, 'Zeitschr. f. Chirurg.,' 1880, S. 138.
17. König.—"Zur Revision der Lehre über die Vornalune grösserer Operat. (Amputationen) bei diabetischen Brand," 'Zeitschr. f. Chir.,' 1887, S. 225.
18. Landouzy.—'L'Union médicale,' 1861, p. 260.
19. Marchal de Calvi.—'Gaz. des Hôpitaux,' 1852, p. 178; 1853, Feb. 5th and July 14th; 'L'Union méd.,' 1861, p. 296.
20. Musset.—'L'Union médicale,' 1856, p. 263; 1857, p. 211; 1859, p. 518; 1861, p. 402.
21. Paget.—'Studies from Old Case-books,' London.
22. Regnier.—"On the Value of the Knee-jerk for Prognosis," 'Bull. Soc. Chirurg.,' 1887, p. 444.
23. Roser.—"Diabetes und Sepsis," 'Deutsch. med. Wochen.,' 1880, Hft. 1 and 2.
24. Saundby.—'Lectures on Diabetes.'
25. Schüller.—"Phlegmon und Gangränos: Processen bei Diabetes," 'Berlin klin. Wochen.,' 1888, SS. 944 and 989.
26. Schuster.—"Zweitälle von Fussgangrän bei Diabetes Mellitus," 'Deutsch. med. Wochen.,' 1888.
27. Sonnenburg.—"Ueber Zellgewebs.-Entzündungen bei Diabetes," 'Berlin. klin. Woch.,' 1882, S. 705.
28. Spencer.—"Amputations in Diabetes," 'Trans. Roy. Med.-Chir. Soc.,' 1892, p. 395.
29. Willett and Walsham.
30. Zeller.—'Würtem. med. Correspondenzblatt,' Bd. lv, Hft. 9.

## Part VI.

### *Mal Perforant.*

We have already mentioned chronic ulcerated conditions (of traumatic or spontaneous origin) as antecedents to the death of one or more toes; the particular form of ulcer known as "mal perforant planteur" or perforating ulcer deserves more particular notice: Tuffier and Chipault,[1] give a very good review of this form of disease from a general and wide point of view; they emphasise the conclusion that the nerve lesion is on the centrifugal side of the neurotrophic arc (at the nerve-roots, on their course, or at their terminations), the most usual one being peripheral neuritis. It is well known that certain nerve lesions occur during the course of diabetes, peripheral neuritis being especially apt to supervene (Ross [2]). In diabetes the knee-jerk is "very commonly" lost (Saundby [3]); this has been considered as a circumstance of importance for prognosis: from the physician's point of view it is thought bad by Ross from his own experience, and also Bouchard, whilst Rosenstein declines to give any weight to it. As a surgeon, Reynier [4] gave his opinion to the Société de Chirurgie of Paris that the prognosis in cases of absent knee-jerk was of the worst sort. It is to be noticed that all the cases upon which he founded his opinion were of an acutely septic nature, which in itself may have had some effect (v. Ross, op. cit.); in the discussion of the question Verneuil remarked that it was a matter of great interest, but that future observation must decide its true worth; he said, further, that at present there was no trustworthy guide to prognosis in the disease, both the amount of sugar and the response to treatment being valueless.

Other signs of nerve degeneration observed are pains (often very severe), and local anæsthesia and hyperæsthesia.

[1] 'Arch. gén. med.,' 1891, t. xxviii, pp. 257 and 413.
[2] 'Treatise on Peripheral Neuritis,' London, 1893.
[3] 'Lectures on Diabetes,' p. 108.
[4] 'Bull. Soc. Chir.,' 1887, p. 444.

Given the proper nerve lesion, Tuffier and Chipault (loc. cit.) analyse the immediate and localising causes into three heads: —(1) Slight traumatism,—recurring pressure may be enough. (2) Chemical agents, hence the difficulty of finding suitable antiseptic applications; they note especially three bad cases of "phlyctenular erythema" caused by the use of iodoform. (3) Infective agents; most often, however, the ulcer passes from an aseptic to an infected condition with the onset of pain and hyperæsthesia in place of no pain and anæsthesia.

Many cases give a history of exposure to cold, or of actual frostbite; or of suppurating hard corns or of hard corneous thickenings of the cuticle, which do not appear to suppurate.

Complete symptoms of tabes dorsalis need not be present; Tuffier and Chipault very reasonably inveigh against the uselessness of endeavouring to make "mal perforant" as it were the "hall mark" of tabes; the unnecessary term "pseudo-tabes" appears to have been coined for the sake of diabetic patients.

Perforating ulcers are naturally liable to be secondarily inoculated with septic organisms of varying virulent power, as occurred, for example, in the case whose history we have already given; we mentioned before that such circumstances might lead to gangrene of the toes. Bowlby,[1] gives a series of four cases in undoubted diabetics where "maux perforants" became actually inflamed, and another case of gangrene of the foot, the prime cause of which is not given; in it peripheral neuritis was found post mortem; two of the cases underwent amputation, in both the flaps sloughed with fatal result, two of the cases not operated upon died rapidly with spread of the septic condition, and in one the result is not stated. He considers that peripheral neuritis plays an important part in the origin of diabetic gangrene, but that it is only to be regarded as a predisposing cause; we cannot but think that he fails to prove that it can be considered as a cause, except in so far as it conduces to the formation of ulcers or "maux perforants," which, quite secondarily, may cause gangrene of the foot or toes.

The sites of the perforating ulcers are, as in other cases,

[1] 'Treatise on Injuries and Diseases of Nerves,' London, 1889.

usually at the "points d'appui" of the feet, viz. the ball of the hallux of the little toe and the heel, all on the plantar surface of the foot, whence the name "mal perforant planteur." Albert [1] records a case commencing as an ulcer close to the nail of the hallux which closely resembled a mal perforant, and he would have named it so "had it not been in the wrong place." This opens the question, at present not answerable, how far the chronic ulcerations about the feet, &c., which occur in diabetes are related to "mal perforant;" it would seem that such an ulcer may become "mal perforant" by the subsequent development of peripheral neuritis, either through the diabetes, or as Tuffier and Chipault,[2] suggest (in non-diabetic cases) by the occurrence of a "septic ascending neuritis."

The perforating ulcer in its typical condition is a round, punched-out lesion, surrounded by a thickened zone of the corneous layer, and around it are areas of anæsthesia, thermanæsthesia, and paræsthesia, though sometimes these symptoms are more extended, *e. g.* in patches on the dorsum of the foot, leg, &c.; probing causes no pain, but reveals that bone or an articulation is exposed.

They are frequently symmetrical in position on the two feet.

In some cases they heal fairly readily. Jeannel[3] remarked the favorable influence of anti-diabetic treatment; in others they are extremely refractory. Tuffier and Chipault's remark concerning the action of chemical antiseptics deserves to be borne in mind: "When they do heal, they nearly always break out again" Jeannel insists on the importance of surgical boots specially designed to prevent pressure upon the cicatrices.

We have been unable to discover any records of cases of operation for "mal perforant," except when acute sepsis has occurred, with mortification about the feet. Tuffier and Chilpault remark that the affection is "medical," and that therefore the treatment should be on "medical" lines.

Gascuel found 14 (= 15 per cent.) diabetic cases

[1] 'Allg. Wien. med. Zeit.,' 1885, S. 1.

[2] Loc. cit. supra.

[3] 'Revue de Chirurgie,' 1886, p. 31.

amongst 91 instances of "mal perforant," but some of these may have been glycosuric only.

Including 4 cases from Bowlby,[1] and 2 from Spencer,[2] we have a total record of 24 cases of perforating ulcer with glycosuria; of these, 16 are apparently true diabetic, whilst in 8 the nature of the glycosuria is uncertain. We append short accounts of the cases thus collected; two only have been recorded in English.

### *Cases of Mal Perforant with Diabetes.*

BERJA.—M. J—, M. Wound in cutting corn, sloughing, glycosuria, treatment, healing in three months. Three years later, same foot crushed; glycosuria again appeared, healing lasted six months. Some time after a "mal perforant" developed spontaneously on the ball of the right hallux. No further history.

DUPLAY.—M. 47. Diabetic (no diabetic history given). In 1883 an ulcer formed under the second toe, which gradually deepened to the bones.

1887. A symmetrical ulcer appeared on the other foot.

1889. Gangrenous condition of left foot and diffuse phlegmon of the right. Incisions were made, and the necrosed first phalanx of the left hallux removed. Patient improved and was discharged.

June 24th, 1890. Returned with both ulcers open, painless, with callous edges; anæsthesia and thermanæsthesia; polyuria and 2 per cent. sugar were then present. No further history.

DUVERNOY.—Obs. I. M. F—, M., 55. Diabetic twelve years, drinks a good deal of alcohol; went to Vichy several times with improvement; during period of confinement to room crateriform ulcer with thick zone of epidermis formed just behind metatarso-phalangeal joint of fifth toe. Anæsthetic zone and paræsthetic zones 1, 2, and 5 cm. respectively; arteries not markedly sclerosed. Diabetic diet and local dressing.

Plaques of cutaneous gangrene occurred on foot and toes: plaque on outer side of foot led to exfoliation of muscle, tendon, &c., and of the first phalanx, fifth toe. Complete cicatrisation occurred in a couple of months. Eventually patient died, four or five months afterwards, of pulmonary tuberculosis.

DUVERNOY.—Obs. II. C—, F., 60. Thirst, polyuria, glycosuria, gangrenous plaque formed and healed close to nail of hallux. Three months later a round ulcer formed on the plantar aspect of the heel; it was surrounded with zone of thick epidermis. No notable atheroma; lung trouble. Three months' healing of ulcer and formation of new one on the outer side of the heel. Continued wasting and increase of cough, with hæmoptysis; died in a few months of syncope.

KIRMISSON.—Obs. I. M., 58. Three years' history of diabetes; "mal perforant" on the sole of right foot; gangrene of first two toes; disarticulation at metatarso-phalangeal joints; absolute anæsthesia was noticed; the hæmorrhage very slight; abscesses occurred afterwards, and the anæsthesia spread over the whole foot.

---

[1] Op. cit.

[2] 'Trans. Med.-Chir. Soc.,' 1892.

KIRMISSON.—Obs. IV. M., 46. (Under Guinard.) Alcoholic; had been under treatment for diabetes; one month's history of pain and swelling in foot; "mal perforant" developed; no symptoms of locomotor ataxy; further history unknown.

KIRMISSON.—Obs. VI. M., 45, stout. Several carbuncles on trunk; "mal perforant" in each foot, having begun six years ago with a corn; œdema of legs; plantar anæsthesia and therm-anæsthesia; no shooting pains. Glycosuria (3·2 per cent.), polyuria, and albuminuria. Carbuncles opened with the thermo-cautery. Three weeks later, erysipelas set in, the glycosuria ceased, and the patient expired on the ninth day.

KIRMISSON.—Obs. VII. M., 58. (Under Verneuil.) "Mal perforant;" glycosuria; two years' thirst, polyuria, and wasting; knee-jerks absent; no other symptoms of locomotor ataxy.

JEANNEL.—Obs. VII. D—, M., 60. Outer edge of sole; thick corneous plaques; similar condition under calcaneum; both appeared two years ago; now sphacelation has occurred; in removing one slough troublesome hæmorrhage was started. Urine, 2 litres, contains a considerable amount of sugar. Transient attack of right hemiplegia two years ago; no other history. Diabetic treatment ordered; improvement followed, and patient was discharged after two months almost healed, with less glycosuria.

ROSS.—J. K—, M., 55. Ten months' history of thirst, polyuria, wasting, and pains in thighs, legs, and feet. Knee-jerks absent, also plantar reflex; tingling and paræsthesia of legs. Deep ulcer surrounded by some congestion (which began as a small swelling which burst and discharged pus) below right external malleolus. Cicatrix of old ulcer beneath metatarso-pharyngeal joint of right hallux.

Urine, 57—84 oz.; sp. gr., 1042—45; sugar, 25—35 gr. per oz.

Under a rigid dietetic treatment the ulcer healed, and the patient improved generally.

## *Cases of "Mal Perforant" with Glycosuria of Doubtful Nature.*

DUVERNOY.—Obs. III. Jewess, F., 60—65. Somewhat obese; hallux swollen and red, at base on sole of foot sinus 1 cm. deep; no bone felt, but small sequestra had been discharged; surrounding paræsthesia; glycosuria ·7 per cent. Patient not seen again.

DUVERNOY.—Obs. IV. M. L—, M., 45. Heavy build, not fat; had been told he had tabes dorsalis; diminished knee-jerk, weakness of legs; no pains, anæsthesia, or local trouble. A year ago had ulcers on feet, which healed slowly. Urine, 3 litres; 1·29 per cent. sugar. No other diabetic symptoms (except loss of teeth). Diet and Vichy water ordered; already improved in a few weeks; in a couple of months the glycosuria ceased. Later on he walked about too much, small ulcers between the toes and superficial gangrene on the sole of the foot resulting. The urine again became glycosuric. Improvement followed with rest and treatment, so that only one insensitive thick-edged ulcer remained after a month. At the end of another three months, including three weeks' rest in bed after a fall downstairs, there was no advance. A variety of dressings tried: iodoform, iodol, phenol, thymol, and "vin aromatique;" none of these seemed to possess any particular advantage.

JEANNEL.—Obs. V. R—, M., 45. Began to suffer from "mal perforant" under the head of the left hallux five years ago on the site of a corn which became

painful and ulcerated, a symmetrical ulcer appearing on the other foot. For four years the ulcer was treated and healed, only to return at frequent intervals. Then the patient had an attack of facial erysipelas, soon after which the ulcers healed entirely ; but in another month there was a recurrence of both. The joints were attacked, and sequestra discharged and were removed. After this the right foot healed, and later on the left also. The right foot eventually gave much anxiety from inflammatory œdema of the leg and foot. Hot carbolic acid (1 per cent.) baths dispersed the swelling, and amputation of the hallux was contemplated, but put off as glycosuria (1·8 per cent.) was discovered. Patient was dieted, and given bromide of potassium and glycerine. In a few days over three weeks, after removal of sequestra, the foot healed up as if by enchantment. He was discharged with specially designed boots, and remained sound when seen a month afterwards.

Jeannel.—Obs. VI. J. P—, M., 61. When thirty-nine, painful red swelling of left foot ascribed to mental shock. A year after return of swelling the first and fourth toes were cast off in an "ainhum-" like manner. Abscesses then appeared on dorsum of foot, and the other three toes atrophied considerably. Multiple "maux perforants" then appeared about the sole, heel, and malleoli. Surrounding each was a zone of anæsthesia. Years after, phlegmon of arm appeared; glycosuria was then found. Anti-diabetic diet with bromide and glycerine ordered; the sugar was reduced to a trace. There was also some thirst, which also abated. In less than a month the ulcers were nearly healed.

Kirmisson.—Obs. II. M., 53. Not alcoholic. Inflamed corn under the ball of the right hallux. Glycosuria well marked. No other symptoms of diabetes. Perforating ulcer developed. First two toes became gangrenous. Amputation was performed in the lower third of the leg. The wound became gangrenous, and the patient died on the eighth day. Post mortem, the arteries were atheromatous, even in their finer branches. Granular fatty degeneration of muscles.

Kirmisson.—Obs. III. M., 47. (Under Verneuil.) Alcoholic. "Mal perforant" under ball of left hallux. Hæmorrhage occurred. Glycosuria was detected on several occasions. Opium was given, and the foot dressed with carbolic dressings. In a month the ulcer was almost healed.

Kirmisson.—Obs. V. M., 58. Eighteen months ago frostbite of feet. Healed in a year. Now has mitral regurgitation and a four-month-old "mal perforant." Glycosuria (1·0 per cent.) present, which disappeared entirely under diabetic treatment, and the ulcer healed.

Willett and Walsham.—T. H—, M., 47. Alcoholic; gangrene of toes on left foot appeared spontaneously; twelve months ago under Mr. Butlin for perforating ulcer in the sole of right foot; anæsthesia of right foot, hyperæsthesia of left foot. Glycosuria (22 gr. per oz.); no other symptoms of diabetes. Optic atrophy. Ordered regulated diet; toe removed with scissors; spread of gangrene. Ether administered and amputation performed at junction of middle and lower thirds of leg; blood-vessels in good condition; sloughing of flaps; spread of gangrene to thigh; death in fourteen days from septic conditions; remained fully conscious.

Heusner records a most interesting case of "mal perforant," accompanied and followed by a series of nerve disturbances, in which glycosuria appeared as a late symptom. A *résumé* of the case has already been given in English by Saundby.

*Bibliography of "Mal Perforant" in Diabetes.*

1. Albert.—'Allgemeine Wiener med. Zeitung,' 1885, S. 1.
2. Berger.—'Bulletin de la Soc. Chirurg.,' 1883, p. 369.
3. Bowlby.—'On Injuries and Diseases of Nerves,' London, 1889.
4. Duplay.—'Union médicale,' 1890, p. 37.
5. Duvernoy.—'Archives générales de Med.,' 1891, t. xxviii, p. 603.
6. Heusner.—'Deutsch. medicin. Wochensch.,' 1885, S. 250.
7. Jeannel.—'Revue de Chirurgie,' 1886, p. 31.
8. Kirmisson.—'Archives générales de Med.,' 1885, t. xv, p. 44.
9. Ross and Bury.—'Treatise on Peripheral Neuritis,' London, 1893.
10. Spencer.—'Trans. Med.-Chir. Soc.,' London, 1892.
11. Tuffier and Chipault.—'Arch. gén. de Med.,' 1891.
12. Willett and Walsham.—'Med. Press and Circular,' 1885, 610.

Lastly, Mr. Durham tells us that he has operated upon diabetic patients in a considerable number of cases of very various nature (*e.g.* from the removal of nasal polypi to the performance of amputation through the thigh), and that, as the result of his experience, he does not consider the existence of diabetes to be seriously deterrent to operative measures in cases in which, on general grounds, such measures are called for. His cases, as a rule, have done well, although healing has been delayed, especially when union by granulation has taken place.

# THE ACCIDENTS OF ANÆSTHESIA.

By GEORGE ROWELL.

(Communicated by the Editors.)

For the sake of convenience, this subject may be considered in the following way; the term "accident" being used in its widest sense, and including any deviation from the normal performance of the functions necessary for life during anæsthesia.

*Accidents especially connected with an Imperfect Degree of Anæsthesia.*

A. The circulation fails.

I. Causation.
1. Fear or fright.
2. Surgical shock.
3. Air-limitation in administration.
4. Deficient oxygenation of the blood due to inefficient respiration.
5. The act of vomiting.
6. A too high percentage of chloroform vapour.

Predisposing Causes.

II. Symptoms. (Characterised by sudden onset.)

III. Treatment: Preventive and remedial.

B. Respiration is impaired.

General Predisposing Conditions.

I. Causation.
1. Voluntary acts of patient.
2. Conditions of muscular contraction, *e. g.* spasm of respiratory muscles.
3. External causes, *e. g.* pressure on chest.
4. Internal obstruction, *e. g.* contraction of internal muscles, venous congestion of mucous membrane.
5. Foreign bodies.

II. Symptoms. (Stertor, cyanosis, &c.)

III. Treatment: Preventive and remedial.

*Accidents which are not especially associated with an Imperfect Anæsthesia.*

C. Failure of the circulation.

I. Causation.
1. Hæmorrhage.
2. Reflex inhibition of the heart.
3. Over-dosage.

II. Symptoms. (More gradual in appearance in 1 and 3.)

III. Treatment.

D. Impairment of respiration.

I. Causation.
1. Foreign bodies.
2. Asphyxia independently existing.
3. Morbid conditions, *e. g.* enlarged thyroid.
4. The effect of operations.
5. Failure of breathing from weakened circulation.
6. The effect of morphia.
7. Over-dose.

II. Symptoms. (In many cases gradual failure of respiration.)

III. Treatment.

*Accidents especially connected with an Imperfect Degree of Anæsthesia.*

A. *The circulation fails.*—Except under rare conditions, this accident only happens in practice when chloroform or a mixture containing chloroform is used.

I. *Causation.*—There are several ways in which it may be brought about:—(1) fear or fright; (2) by the shock of some surgical condition or proceeding; (3) by air-limitation in administration; (4) by deficient oxygenation of the blood,

due to inefficient respiration from any cause; (5) by the act of vomiting; and (6) by a too high percentage of chloroform vapour, causing a sudden over-dose.

It has been suggested that stoppage of the heart's action in an early stage of the administration may arise from the reflex effect of chloroform vapour upon the mucous membrane of the air-passages. I am unacquainted with either clinical or physiological evidence to support this idea. Another supposed cause of early heart failure is put forward by Dr. Robert Kirk.[1] He states that during the early part of the inhalation, when the lungs are full of chloroform vapour, but before any is absorbed, the sudden withdrawal of the chloroform may produce a profound change in the circulation, culminating in syncope. The facts adduced in support of this theory are meagre, and are often explicable, as was suggested by Sir J. Lister,[2] in another well-known way, as they apply equally well to the theory that the act of vomiting was imminent (see p. 440).

1. *Fear or fright.*—Not only does excessive terror predispose to accidents of this class by, presumably, weakening the heart's action and producing irregularities of respiration, and so lessening the resisting power of the patient to any unfavorable event that may happen; but it has also been the undoubted cause of several deaths from syncope. Snow recorded the case[3] of a pale delicate woman, aged 37, upon whom Mr. Cæsar Hawkins was about to operate for the removal of a glandular tumour of the breast. She was very nervous on entering the theatre. Owing to the excitement from which she suffered, she did not inhale the chloroform vapour, which was very dilute, in a satisfactory manner. Her breathing was very irregular. She was then begged to control herself, and to breathe more quietly. This had no effect upon the character of the breathing, and suddenly, "not more at the most than one and a half minutes from the beginning of the inhalation," the breathing suddenly ceased, and she became "deathly pale." Cold water was dashed upon the face and chest, and artificial

[1] Kirk, 'A New Theory of Chloroform Syncope.'
[2] Lister, 'System of Surgery,' vol. iii, p. 618.
[3] Snow, 'Anæsthetics,' p. 209.

respiration was resorted to, but with no effect. No pathological changes were found in the viscera except slight fatty degeneration of the heart. Snow speaks positively of the small quantity of chloroform which was inhaled, and he tested the blood for chloroform with a negative result. Several other deaths of a similar nature are on record,[1] in some of which no anæsthetic was used. It is not difficult to understand how fear can kill, when we think of an ignorant or superstitious person, who knows that deaths have happened in the state into which he is going, and who exaggerates the already sufficiently unpleasant subjective sensations of the first stage of anæsthesia. Mr. W. E. Henley's description of these sensations, in 'A Book of Verses,' is of none the less value as a true observation because of its poetic power.

. . . . "Then the lights grow fast and furious,
And you hear a noise of waters,
And you wrestle, blind and dizzy,
In an agony of effort,

"Till a sudden lull accepts you,
And you sound an utter darkness. . . . ."

He cannot be a good anæsthetist who has no sympathetic appreciation of his patient's feelings.

2. *Shock.*—Direct reflex inhibition of the heart will be considered later, because it is not clinically so clearly connected with too light an anæsthesia. The shock here alluded to is a more general condition, and has been a favourite explanation of deaths occurring in imperfect degrees of anæsthesia, but I believe its danger to life has been overestimated. Deaths are recorded[2] which admit of no other explanation, but it is open to question whether they would not equally have happened without the anæsthetic. In illustration I may quote the case[3] of a boy, aged 14, who was given chloroform by the open method for the purpose of reducing a dislocated elbow. He was pale and

[1] Snow, op. cit., pp. 201, 203, 245; Lister, op. cit., p. 600; 'Brit. Med. Journ.,' 1877, vol. ii, p. 692.

[2] Lister, op. cit., p. 600.

[3] 'Lancet,' 1882, vol. ii, p. 153.

trembling, but he took the anæsthetic well, and his pulse and respiration were good. After five minutes he was thought to be well under, and the dislocation was reduced without difficulty. He groaned and kicked whilst this was being done, and no more chloroform was given. A few minutes later his breathing had become slow, and he suddenly turned deadly pale. The heart's impulse was imperceptible. His head was lowered, cold water was applied to the chest, artificial respiration was kept up, ammonia was applied to the nostrils, and the battery was used. He is stated to have been dead two minutes after the onset of the symptoms. As a cause of death shock is often associated with mental emotion. Snow quotes[1] from Simpson a death at Edinburgh immediately happening when an abscess was lanced. The case was considered too trivial for an anæsthetic. Snow, the extent and value of whose work becomes more apparent as our knowledge increases, pointed out that the paralysis of the heart produced by poisonous doses of chloroform is pathologically a relaxation of that organ, with the cavities full of blood.[2] The syncope of pain or mental emotion is an affection of the nervous system. It is accompanied by vaso-motor changes, and the heart probably stops in large measure because of a deficiency in the amount of venous blood returned. It has often been found empty after death. Now the action of chloroform, except in excessive doses, is, as we shall see, to stimulate the vaso-motor centre. Hence chloroform, so far from predisposing to shock, even in conditions of imperfect anæsthesia, is actually a slight protection from this accident. When consciousness and the reflexes are completely abolished, syncope from fear and from shock seems impossible. In the imperfect degree of anæsthesia that we are now considering, the shock produced by pain, or by interference with parts closely connected with the sensory mechanism, has a most undoubted influence upon the pulse.[3] But, except in the case of very powerful stimuli, such as are described on p. 456, and during a sufficiently light anæsthesia, the circulation

[1] Snow, op. cit., p. 245.

[2] Ibid., p. 222.

[3] Bickersteth, 'Monthly Journal of Medical Science,' Sept., 1853.

tends to become good again immediately the cause of shock is removed, and often before that. The fact, however, that deaths have been caused by shock must not be forgotten. A very clear instance of this accident, in which death was averted by inversion and artificial respiration, is recorded by Sir J. Lister.[1] A middle-aged lady was not fully under chloroform when the operation, which was for fissure of the anus, was commenced. She at once ceased to breathe and became deadly pale. The means above mentioned were instantly taken to restore the circulation, with success.

3. *Air-limitation in administration.*—Apart from the possibility of the ingestion of chloroform vapour in a dangerously high degree of concentration, to be afterwards discussed, the liability of air-limitation to cause failure of the heart during the induction of narcosis is well known. This form of accident is little likely to arise with the present methods of exhibiting chloroform. Ether, however, is now almost universally given by the so-called "close method." In the early stages of the administration the exclusion of air is most needed. Occasionally, though practically always in patients who were suffering at the time from serious disease or exhaustion, the heart has failed under ether from this cause.[2] An anæmic and feeble patient,[3] aged 45, was about to be operated upon for an abdominal tumour. Less than half an ounce of ether was poured upon the sponge of an Ormsby's inhaler, which was then applied to the face. The air-valve was open for the first three minutes, and had "certainly not been closed for more than two minutes" when the pulse disappeared. The breathing was still regular and deep. The pupils rapidly dilated. The inhaler was at once removed and artificial respiration and inversion resorted to, but "we got nothing more from the patient than one groan," the report says. No incision had been made. The only noticeable feature at the necropsy was the small size of the heart. After anæsthesia has become well established, no patient under ether is likely to need a sufficient

[1] Lister, op. cit., p. 623.

[2] Mills, 'Brit. Med. Journ.,' 1881, vol. i, p. 103; and 'Lancet,' 1887, vol. ii, p. 1132.

[3] Tait, 'Brit. Med. Journ.,' 1882, vol. ii, p. 103.

deprivation of oxygen to produce heart-failure, and with only ordinary care it will not be likely to happen in good anæsthesia.

4. *Deficient oxygenation of the blood from inefficient respiration.*—The various means by which the proper performance of the act of respiration is checked will be considered below (p. 448). Apart from the aid to the circulation which respiration mechanically affords, cardiac action is prejudicially affected by a deficiency of oxygen in the blood. In the stage of excitement, when these derangements of respiration are most prone to happen, depression of the circulation is often noticeable. Sudden syncope from this cause has brought about many deaths.[1] The following is an example of this mode of death :—A man,[2] aged 31, took chloroform in order that a tumour of the forehead might be removed. It was administered by the open method upon a towel. After one or two minutes more chloroform was added to the towel, and violent struggling at once occurred. The face became livid because of the inefficient respiration during the struggling. The administration was stopped, and the breathing was noticed to be very laboured. Suddenly "a change in the face" was observed, and the patient gasped and died. Treatment was of no avail. Sometimes the heart fails more gradually. In either case, death may occur from heart-failure before the asphyxial condition kills. The accident is most to be feared in excitable, muscular, and alcoholic people, who may become very rigid, and hold their breath for long periods. Dr. F. Hewitt has called attention to this point,[3] and mentions as an illustration that, in feeble old persons, such slight interference with breathing as occurs when a mouth-gag is inserted often produces an immediate effect upon the pulse, which effect ceases as soon as the impediment to breathing is removed by pushing forward the lower jaw.

5. *Vomiting.*—Depression of the circulation is often the first warning that vomiting is imminent. There is reason

[1] 'Lancet,' 1881, vol. ii, p. 430; and 'Brit. Med. Journ.,' 1888, vol. ii, p. 719; and 1883, vol. i, p. 351.

[2] 'Brit. Med. Journ.,' 1881, vol. i, p. 62.

[3] Hewitt, 'Med.-Chir. Trans.,' vol. lxxiv, pp. 119, 120.

to believe that some of the deaths that have occurred from primary syncope, under chloroform, have been caused in this way, before vomiting has actually taken place. I well remember the case of a lady in the country, to whom I was administering chloroform, some years ago, at a too cautious rate. Sudden symptoms of syncope of an alarming character appeared after two or three minutes' inhalation. Before one had time to apply means of treatment the occurrence of violent vomiting put an end to the condition, and the inhalation was continued without an abnormal symptom. Syncope may occur with, or immediately after, the act of vomiting, and terminate fatally. A gentleman,[1] aged 66, took ether for colotomy, to relieve obstruction of the bowel. He had no complicating disease. Nothing solid had been taken for some days. The ether was given by means of Clover's inhaler. The patient made no resistance, took it well, and in two minutes was "well under." His pulse was at this time strong, when he seemed inclined to vomit, and shortly afterwards a brownish fluid was ejected. He took one inspiration after this, and then his head sank back and he quietly died. There was no other respiratory effort, except one short gasp during the performance of artificial respiration. Every effort to restore him was useless. It was quite clear that there was no mechanical obstruction to breathing, and little more than an ounce of ether was used. In many cases of this kind the question will arise whether the syncope be not due to obstructed breathing. The completion of the act of vomiting is usually a remarkably strong stimulus to the circulation, causing the colour to immediately return, and the pulse to become full and strong.

6. *The effects of a sudden over-dose of chloroform, produced by a too high percentage of the vapour in the inspired air.*—By careful experiments Snow clearly showed that the sudden intake of a strong vapour may cause instant death by paralysing the heart.[2] Sir J. Lister observed this form of death in a donkey.[3] M. Paul Bert also found this to occur experimentally. The Hyderabad Commission

[1] 'Lancet,' 1880, vol. ii, p. 376.
[2] Snow, op. cit. (Experiments).
[3] Lister, op. cit., p. 601.

interpreted their results in such a way as to minimise the direct effect of chloroform upon the heart, but the labours of Snow, who found that instant cessation of movement was caused by blowing chloroform vapour upon an exposed heart; of Dr. MacWilliam,[1] and of Drs. Gaskell and Shore[2] have established beyond doubt the physiological fact, which had long been suspected from clinical observation, that chloroform is a direct poison to the heart muscle. Many deaths have undoubtedly happened during imperfect anæsthesia from this sudden over-dose.[3] The manner in which the accident is most likely to come about is as follows:—In the stage of struggling the breath is often held. Chloroform vapour, being more than four times as heavy as air, will become concentrated under the towel or mask during this time if the latter is kept closely applied, which it might be from a desire to hasten the passage of the patient through this troublesome stage. A long inspiration follows: the patient dies. It should be borne in mind that the heart which fails in this way may be already weakened from the cessation of breathing, which fact would therefore predispose to the accident.

The following is a characteristic example of this mode of death:[4]—A healthy man, aged 26, was admitted to the Glasgow Infirmary in 1884 for a simple fracture of both legs. To set the fractures chloroform was administered. There was no nervousness. The anæsthetic was exhibited by the open method, and as soon as the inhaler was applied to the face, the patient took several rapid shallow breaths, as if anxious to quickly become unconscious. Following these were three or four deep inspirations. At this point the inhaler was removed, as it was felt that a large quantity of chloroform must have been inhaled. The patient was noticed to have his eyes wide open, his pupils dilated, and no conjunctival reflex. The pulse had disappeared, but the breathing still continued, and the face was still a fairly good colour. Vigorous treatment, by partial inversion,

[1] MacWilliam, 'Brit. Med. Journ.,' 1890, vol. ii, pp. 831, 890, 948.
[2] Gaskell and Shore, 'Brit. Med. Journ.,' 1893, vol. i, pp. 105, 164, 222.
[3] 'Brit. Med. Journ.,' 1883, vol. i, p. 351.
[4] Ibid., 1884, vol. ii, p. 811.

artificial respiration, ether subcutaneously, and galvanism, was at once applied, but the patient was dead, and the heart does not seem to have acted again.

As is seen by a consideration of the foregoing remarks, failure of the circulation during states of imperfect anæsthesia is often brought about by a combination of two or more of the causes described. There are also certain conditions which act as predisposing causes. Chief amongst these are enfeebled and diseased states of the heart, conditions of exhaustion and collapse, and idiosyncrasy. Valvular disease has little, if any, predisposing effect, as compensation by hypertrophy is usually present. There are grounds for believing that fatty degeneration of the heart gives a stronger predisposition. Exhaustion and collapse, by directly enfeebling the circulation, are more important, and may require consideration and treatment. Much has been attributed to idiosyncrasy by Billroth, Snow, and others. It is very unfrequently that one meets with any marked instance of excessive susceptibility to a particular anæsthetic in practice, although patients vary considerably in this respect. A case that occurred in my practice is described on p. 461. Constant watchfulness would probably discover the fact in time to avert harm.

II. *Symptoms.*—The most characteristic feature of these symptoms is their sudden onset. Even when hampered breathing is the cause of the syncope, this may be noiseless, and consequently overlooked. The first symptom which attracts attention is usually pallor. When the accident is caused by an asphyxial condition this pallor will be associated with lividity. The pupils suddenly dilate widely : the pulse becomes feeble and fluttering : the heart may stop beating. In some conditions pulse-failure may occur whilst the breathing is still good : therefore the administrator should keep himself acquainted with the character of the pulse. The weakening of the heart's action, which chloroform is now known to produce, is disguised by the stimulating effect of the agent on the vaso-motor centre, so that the pulse, when felt a moment before the accident happens, may be good. *In chloroform narcosis the strength of the pulse is a very small indication of the condition of the*

*heart.* The frequent occurrence of swallowing may point to the advent of an attack of vomiting.

III. *Treatment.*—Owing to the sudden manner in which failure of the circulation usually occurs, when associated with too light an anæsthesia, preventive treatment is here all-important. No undue chilling of the patient should be allowed, and he should be as nearly as possible in a horizontal position. The presence of terror should always be noticed, and all possible means taken to prevent and allay it. It is not necessary to add that quietude should be observed until the patient is unconscious, and that the operation should never be commenced until complete anæsthesia has been attained. The advisability of administering stimulants should be considered in cases of collapse, &c., before the operation. Mr. C. Heath [1] recommends in such cases the injection of two ounces of brandy with four of hot water *per rectum.* This undoubtedly has a marked effect in exhaustion, or shock from injury, although, as we shall see, it is worse than useless as a means of stimulating a chloroform heart.

Fortunately the class of cases dependent upon deficient oxygenation of the blood are usually capable of prevention. A free supply of fresh air must always be obtainable in chloroform administration. The respiration must be carefully watched, and any obstructive or embarrassed condition at once treated as will be described below (p. 452). When ether is given to weakly patients a due supply of fresh air must be allowed (p. 438), to the extent of giving it in extreme conditions of exhaustion and of disease by the open method. The occurrence of vomiting may be guarded against by ensuring an empty stomach and taking care that the bowels have acted. Vomiting is perhaps more likely to occur in imperfect anæsthesia under ether than under chloroform; but as ether is nowadays mostly preceded by nitrous oxide, or by chloroform, or the A. C. E. mixture, it is rarely seen. When it does happen there is little or no tendency to depression of the circulation. By attention to the points mentioned, and by a properly continuous and free adminis-

[1] Heath, 'Brit. Med. Journ.,' 1889, vol. i, p. 234.

tration, vomiting should practically never take place until after the operation.

The intake of an unduly strong chloroform vapour should be most carefully and constantly guarded against. When the breath is held, the inhaler should always be removed a slight distance from the face, in anticipation of the deep inspiration which shortly follows. We are indebted to Sir J. Lister[1] for showing that the amount of chloroform given off by the open method as now practised is only 4·5 per cent., and of this only part is inhaled. The administration of chloroform should be free and continuous until good narcosis is induced. If the state of anæsthesia during the operation is allowed to become too light, as will be seen by a slightly dilating pupil and returning reflexes, the patient will again become liable to the accidents we are discussing. Consequently when he is once properly anæsthetised he should be kept so until the conclusion of the operation. The proper selection of the anæsthetic agent employed is an important point as a preventive measure.

It will now be convenient to discuss the general treatment of syncope, however produced. Where this is possible, the first indication is naturally to remove the cause. The anæsthetic should be immediately withheld, and treatment applied with a view of restoring the circulation. The head should be lowered. According to Dr. F. Hewitt, and as I have also seen, rubbing the lips and cheek with a rough towel often has a marked effect in restoring the colour and pulse in the lesser degrees of faintness. Cold water to the face and chest may be a useful adjunct. Placing the patient upon his left side is also of use. *In all cases of syncope, the proper maintenance of respiration is of the first and last importance*, and whenever the respiration is failing it should be assisted artificially. No time should be wasted in the application of other treatment at the expense of this. Bearing this point in mind throughout, the treatment should be directed to stimulate the heart. The most practical, and one of the most beneficial means by which this can be attained is by Nélaton's method of inversion of the patient. Concerning the *modus operandi* of this measure, Sir J

[1] Lister, op. cit.

Lister found[1] that the raising of any part of the body causes contraction of the arteries in that part through the vaso-motor system, and that the converse also obtains. It seems to me that the heart in inversion of the patient is clearly subjected to an increased external pressure, since it is under the weight of the loose abdominal contents, and I believe that this fact helps to explain the stimulating effects of inversion. Dr. Kelly's method of inversion and artificial respiration combined[2] is stated to have proved effectual in resuscitating all the cases (twelve in number) in which it was employed. An assistant jumps upon the operating table between the patient's feet, and with his arms under the knees elevates the body so that the buttocks are raised to the height of a foot from the table. The head is extended over the top of the table, and the administrator, supporting the head against his thigh, performs artificial respiration with one hand on each side of the lower ribs, pulling them forward and compressing alternately. It may be here mentioned that it is useful to get the hand under the lower left ribs, as pressure upon the heart excites contraction. With regard to drugs, the researches of Wood[3] are worthy of the most careful consideration. Brandy, although of use in surgical shock and collapse, he found to have a depressing effect in chloroform syncope. Ammonia and amyl nitrite he showed to be useless in improving the heart's beat. But he found that the subcutaneous injection of small doses of digitalis had a marked and uniform effect in this direction. When drugs are used, then, for the condition we are considering, five minims of the tincture of digitalis hypodermically should be administered. I believe that I have seen the subcutaneous injection of ether improve the pulse. I can speak more strongly, from experience, of the powerful effect of a hot sponge to the perinæum in cases of failing circulation. When the condition is dependent upon loss of blood the injection of saline solution, so successfully re-introduced by Mr. Lane, should be resorted to. It remains to consider direct stimulation of the heart. Acupuncture has

[1] 'Brit. Med. Journ.,' 1879, June 21.
[2] 'Brit. Med. Journ.,' 1892, vol. ii, p. 937.
[3] Wood, 'Trans. Berlin Congress,' 1890, vol. i.

very little effect, and is, I believe, now never practised. The effect of the interrupted current upon the heart, it should be remembered, is to produce single contractions and not tetanus. Snow, and many observers since his time, have tried this means, with no very striking results. Dr. MacWilliam[1] has investigated the subject, and finds that induction-shocks to the apex improve the force of the heart's beat. One pole should be placed at the level of the fourth dorsal vertebra behind, and one on the apex-beat.

When an animal's heart is brought to a standstill in asphyxia, a ready method of restoring the circulation is by opening the thorax and squeezing the heart between the thumb and forefinger slightly about 200 times in the minute. Although there is no immediate contraction of the dilated and apparently dead heart, the pulse can at once be felt to beat in the femoral artery with each squeeze. In a short time, if the respiration be artificially restored, the heart will once more beat in a normal manner. It is by no means necessary to empty the heart with each squeeze. A slight pressure, the valves acting automatically, will drive on some blood. With a heart which has ceased to beat from the combined effects of chloroform paralysis, and deprivation of air, this method also answers perfectly well, although more time naturally elapses before the heart's action is restored. I have seen animals to all intents and purposes dead brought round in this way. These facts were pointed out to me by Dr. Starling, who suggested that, other means having failed, the circulation might be kept up in this way in chloroform syncope, when air could be made to enter and leave the chest. I would strongly urge that, should all means of resuscitating a case of chloroform syncope have failed, and always provided that respiration can be artificially performed (see p. 448), the following procedure can do no harm, and may save life :—The skin might be drawn forcibly upwards, and an incision about three inches long made in an intercostal space, just external to the apex-beat. This might be carried through into the pleural cavity, just large enough to admit the first two fingers of the surgeon's right hand, the chest being kept in a state of expiration until the

[1] MacWilliam, 'Brit. Med. Journ.,' 1889, vol. i, p. 349.

fingers were inside. By flexion of the fingers upon the heart, as strongly as possible, and repeated about one hundred times in the minute, *artificial circulation* would be performed, artificial respiration being kept up meanwhile. If only a drachm of blood were driven out of the heart by each squeeze, I think there are strong physiological grounds for believing that after a short time, even though the heart's action had been suspended for three or four minutes, the surgeon would feel it contracting under his fingers, and it would shortly proceed of its own accord. Many of the cases recorded have occurred in strong adults. In a case in which there is no room between the ribs for the surgeon's fingers, the abdomen might be opened, under the heart, and the heart squeezed, through the diaphragm, by the fingers, against the front wall of the chest.

It must be borne in mind that by artificial circulation and respiration the chloroform in the blood would soon be eliminated. I admit that the operation would not be easy to perform unexpectedly. Should recovery occur the dangers of air in the pleural cavity, and of after-suppuration, would have to be reckoned with. Supposing the surgeon's fingers to be aseptic, I do not see why there should be any great risk of the last complication. However, the occurrence of chloroform syncope ought, with our present knowledge, to be an event of extreme rarity; and to prevent is better than to cure.

Should recovery from syncope be tardy, and should relapses occur, hot bottles to the feet, bandaging the limbs, sinapisms to the calves, rubbing the gums with brandy, and hot injections of brandy and water into the rectum, will be likely to help.

B. *Respiration is impaired.*—Accidents of this class, unlike failure of the circulation, occur with quite as great frequency under ether as when chloroform is the agent used. The chief causes which predispose to derangements of the function of respiration in anæsthesia fall under two heads—the type of the patient, and the existence of certain diseases. Fat and flabby people appear to be peculiarly bad subjects, and so are the plethoric and thick-necked. Alcoholic and muscular persons are prone to much excitement, and very

nervous patients usually give some trouble. Of conditions of actual disease, ascites, fluid in the pleural cavity, and emphysema will usually make the case an anxious one, and tumours and aneurisms which press upon any part of the air-passages have an important influence. The whole subject of respiration during anæsthesia has been ably and exhaustively treated by Dr. F. Hewitt.[1]

I. *Causation.*—We are still considering states of imperfect anæsthesia, and it appears that impairment of respiration is likely to occur from (1) voluntary acts of the patient; (2) conditions of muscular contraction; (3) external causes; (4) any obstruction within the air-passages not included under the fifth class, which is (5) obstruction due to foreign bodies.

1. *Voluntary acts of the patient.*—As a rule the trouble produced in this way is to be overcome by tact and the anæsthetic. Mr. Watson Cheyne has told me of a remarkable case that he once had of holding the breath. The patient was an officer, who took chloroform a large number of times. On each occasion, just before losing consciousness, he held his breath. When the anæsthetic was removed he became conscious again, but would not inspire any of the chloroform vapour. It was found that the only way he could be tided over this difficulty was by artificial respiration, and on each occasion this was consequently performed while the mask was held to the face. Once unconscious, his behaviour was normal. It seems likely that this condition, as well as conditions of voluntary rigidity, may lead on to the cause of respiratory obstruction next to be described.

2. *Conditions of muscular contraction.*—Struggling is always associated with abnormal breathing, and when it passes into tonic spasm with held breath the condition threatens to become grave. Probably the worst, because the most untractable accident that can occur in anæsthesia is a condition of extremely strong tonic spasm of the respiratory muscles with total cessation of breathing, and in which no air can be made to enter and leave the chest. Sir J. Lister records such a case under chloroform.[2]

[1] Hewitt, 'Med.-Chir. Trans.,' vol. lxxiv.

[2] Lister, op. cit., p. 615.

Mr. Holmes publishes a case which occurred under ether.[1] In each case artificial respiration, even after tracheotomy, had no effect, and the heart continued to act for a considerable period. I am acquainted with the details of a third case (fortunately this dreadful accident is very rare), in which exactly the same course of events took place, ether being the anæsthetic used. Suddenly, in imperfect anæsthesia, the spasm was noticed, artificial respiration proved of no avail, tracheotomy was at once performed, inflation of the lungs was attempted, the heart gradually gave out, and death happened. The pathology of these cases is unknown. What the conditions are that produce rigidity during anæsthesia we have yet to learn. Deficient oxygenation of the blood would seem to play a part; but if this is the principal cause, why is not the accident more frequent?

3. *External causes.*—Amongst these may be mentioned pressure upon the throat, chest, or abdomen; smothering; and alterations in the patient's position, such as when he is placed upon his face, or upon the sound side of his chest when one side is to be operated on, or when the head becomes flexed upon the neck so as to produce an internal obstruction. It should be borne in mind that the breathing of anæsthesia is often almost entirely abdominal, and hence the abdomen should be as carefully guarded from external pressure as the thorax.

4. *Internal obstruction of the air-passages, excluding that due to foreign bodies.*—This, in its different degrees, is the most frequent accident that the anæsthetist has to encounter. It may be produced by altered relations of parts due to muscular action, as when the glottis closes from too strong a vapour, or the larynx is drawn upwards and forwards *en masse*, as in the obstruction of nitrous oxide narcosis (Hewitt); by swelling of the tongue or mucous membrane about the glottis; by falling back of the tongue when the patient is quite flaccid and on his back, in which case its contact with the posterior pharyngeal wall obstructs the channel; by flexion of the head, when the epiglottis is the cause of the obstruction; by half-performed deglutition,

[1] Holmes, 'Brit. Med. Journ.,' 1884, vol. i, p. 508.

when the epiglottis remains on the glottis ;[1] by flaccidity of the lips, tonsils, or soft palate. The majority of these events are especially likely to happen in imperfect anæsthesia. The minor degrees of many of them should perhaps rather be termed incidents than accidents. Many of them are the result of reflex action, as when crowing inspiratory sound occurs in response to surgical interference with the cervix or rectum. I have also observed it in a marked degree in a case of removal of a toe-nail. This particular form I believe to be due to vibrations of the true vocal cords, which are apparently approximated, from the character of the sound, and from the fact that I have seen, in my own larynx, the vocal cords together, when making a noise as nearly alike the crowing sound mentioned as it was possible to produce. Reflexes causing serious obstruction may be also set up by the presence of a small foreign body, such as an accumulation of mucus near the glottis. In deeper degrees of anæsthesia, obstructed breathing from muscular causes cannot arise. The researches of Messrs. Horsley and Semon[2] into the degrees of excitability of the intrinsic laryngeal muscles, under ether, showed that the adductors lost the capability of being made to contract before the abductors ; and they point out that this might be expected by analogy, as the adductors are mainly voluntary for the production of the voice. If, then, laryngeal sound appears during deep anæsthesia, it would seem that the explanation is rather that it is due to flaccidity of the folds of mucous membrane about the glottis. Swelling of parts is especially noticeable under ether, when a certain amount of cyanosis of the face is present. Under nitrous oxide, too, the same feature is often most marked. Many of the causes described act in combination in producing obstruction. Fortunately, although the variety of forms of internal obstruction is considerable, yet with experience and care, any serious departure from the normal course of symptoms rarely occurs ; it can often be foreseen and prevented, and when it does happen, it is nearly always capable of being controlled before the patient's condition becomes dangerous. A typical death

[1] Hewitt, op. cit., p. 114.

[2] Horsley and Semon, 'Brit. Med. Journ.,' 1886, vol. ii, p. 405.

from internal obstruction is recorded by Snow.[1] A rather bloated man, aged 39, inhaled chloroform for an operation on the bladder. He is stated to have commenced to "snore with a peculiar and very profound stertor" when the catheter was passing. His face became congested, and inspirations were drawn at rather prolonged intervals. Methods of resuscitation were used, but the respiration gradually ceased. Artificial respiration with the tongue drawn forwards was vigorously kept up, and the patient's colour began to return, and some effort at inspiration was noticed, when the heart failed and he died. This is also a clear instance of the cardiac failure described on p. 439.

5. With regard to *foreign bodies* it may be said that, whereas obstruction from vomited material, or from such an accidental foreign body as a plate of artificial teeth, is more likely to arise in imperfect anæsthesia, when movement and vomiting are possible, yet there is no general connection of obstruction due to the presence of a foreign body with such a degree of anæsthesia, and this causation of obstruction will therefore be considered later (p. 457). The dangerous accident, which should never be possible, of liquid chloroform obtaining entrance to the throat must be alluded to. In such a case, strong spasm of the glottis is produced, and deaths have rapidly supervened when this calamity has happened.

II. *Symptoms.*—The best recognised feature of obstruction is an unusual degree of stertor. Much might be quoted concerning its many different varieties, but considering that when it calls for treatment the obstruction can, as a rule, be remedied before the diagnosis of the exact form of stertor would be made, the subject, although of great interest, is of small practical use here. Gentle snoring is a symptom of good chloroform anæsthesia, and with ether some stertor is mostly present throughout. *Often there is no noise with the obstruction,* as when the glottis is tightly closed, the tongue "swallowed," or the larynx drawn up under the epiglottis. This is a most important fact, as chest movements proceed, and the condition might be, and it is to be feared has been, overlooked until too late.

[1] Snow, op. cit., p. 182.

Cyanosis increasing in depth is the surest indication that the patient is not receiving enough oxygen, while it also shows that the heart is acting. When the heart fails the face becomes pale, and this blanching may co-exist with lividity, the venous system being still engorged, while the arterial twigs are empty. Should, then, pallor accompany or replace the cyanosis, the condition is urgent indeed : the heart is failing as described on p. 439. Speaking generally, impairment of breathing is not likely to be overlooked if the anæsthetist is attentive to his patient. Clonic movements (jactitation) may occur as a late symptom of obstruction to breathing, when the asphyxial element in the patient's condition preponderates.

III. *Treatment.*—A knowledge of the causation of the impeded breathing is the first essential for its successful treatment. As preventive measures may be mentioned the careful selection of the anæsthetic, and of the means by which it is administered, in morbid conditions ;[1] the free supply of fresh air to avoid undue congestion and swelling of the mucous membrane ; keeping the patient's head on the side ; and the careful avoidance of external pressure upon the thorax or abdomen. This last precaution is most likely to be overlooked when restraining struggling. The anæsthetist should constantly keep himself informed of the continuance of breathing in all stages of anæsthesia. It is not sufficient, as has been seen, to depend upon the movements of the chest and abdomen in respiration ; *if the breathing cannot be heard (or seen distending the bag when the close method is used), it should be felt.* When the breathing once becomes impaired it is not necessary to say that means should at once be taken to relieve the cause of impairment before it becomes worse, and especially when chloroform is in use, as then there is the added danger of heart failure in an especial degree. Internal obstruction due to the epiglottis is relieved by pushing or pulling the lower jaw forcibly forwards, or by extending the head upon the neck. This also relieves the obstruction caused by the half-performed act of deglutition, and, without any such displacement of parts, it is obvious that when the back of the tongue,

[1] Hewitt, 'Practitioner,' 1887, p. 93.

fauces, and soft palate are congested and swollen, this measure will do much to relieve the obstruction. Indeed, many cases of stertor, of which the cause is not at first sight clear, will at once respond to this treatment by a much lessened and softened form of stertor. For spasm about the glottis, forcible tongue traction is the indication, as it is in falling back of the tongue; and in these two simple methods we have the means of curing the majority of cases of the class we are considering. Sir J. Lister first drew attention to these facts,[1] and they are now generally recognised. The most frequent cause of obstruction under chloroform he found to be an approximation of the arytæno-epiglottidean folds, particularly their posterior parts, while the whole larynx was drawn somewhat forwards and upwards under the epiglottis. Forcible tongue traction, while it does not move the epiglottis, causes the glottis to recede, probably, as Sir J. Lister suggested, by a reflex action. The crowing breathing to which allusion has been made may not be relieved entirely by the two means mentioned. Rigidity or spasm is to be treated by pushing the anæsthetic. When rigidity persists under ether, a little chloroform may be substituted. The rigidity will probably soon disappear, and ether may be resumed. When the obstruction cannot be overcome, intubation of the larynx, which might serve perfectly well in certain cases of spasm, or tracheotomy should be at once performed, and artificial respiration, by Silvester's method, should be resorted to whenever and directly it is needed. As an uncommon instance of dangerous obstructed breathing I recall the case of a young woman in Charity Ward, a patient of Mr. Howse, to whom I gave chloroform when House Surgeon in 1887. She was suffering from a sarcomatous growth affecting the cervical vertebræ, which fixed her neck. The anæsthetic was given as she lay on her back. When she had inhaled the chloroform for a few minutes stertor appeared, almost immediately followed by complete obstruction. The usual means of relief proving ineffectual, mainly from her disease, which kept the head rather in a flexed position, and her face, which was by this time much cyanosed, becoming suddenly

[1] Lister, op. cit.

pale, Mr. Howse performed tracheotomy, and she at once recovered without artificial respiration, but the operation was abandoned. Dr. F. Hewitt records a similar case under nitrous oxide. It remains to discuss the treatment of the condition of spasmodic fixture of the respiratory apparatus when artificial respiration and tracheotomy have failed as means of inducing air to enter and leave the lungs. Two means, in the present state of our knowledge, offer a possibility of relief—electrical stimulation of the diaphragm, and the forcible introduction of oxygen into the lungs. There is, as yet, no other way to get oxygen into the blood with sufficient rapidity to be of any use. Dr. F. Hewitt about a year ago conceived the idea that possibly oxygen might be introduced into the system by distending the intestines with it, *per rectum;* and by the kindness of Dr. Starling we were enabled to see that this plan is in practice absolutely useless. The best means of introducing oxygen in such a case would be, I think, to introduce a Trendelenburg's tube into the trachea, blow up the collar, and attach the tube from the oxygen bag to the mouth of the tracheal tube. By considerable pressure upon the oxygen bag, alternating with relaxation, during which the bag might be stretched in four directions by two pairs of hands so as to create a suction upon the air in the chest, it looks on the face of it as if one ought to be able to supply enough oxygen to carry the patient through the emergency. In these cases of spasm the patient's chest is said to be fixed in expiration. If so, one ought to be able to cause the diaphragm to contract by electricity. If the chest is fixed in inspiration, surely it should be possible to press air out of the lungs by compression of the abdomen, applied, for example, by means of a binder hastily slipped round the body. This might be further aided by causing the abdominal muscles to contract with strong induction shocks. Erb, in 'Ziemssen's Cyclopædia,' states that the best way to produce diaphragm contractions is to place one pole at the back of the neck and the other over the front attachments of the diaphragm. Dr. Washbourn was kind enough to try for me several ways of accomplishing this in the electrical department. We obtained very good contractions of the

diaphragm when the two poles of an interrupted current were placed on the lowest costal cartilages, one on each side, and each about one and a half or two inches from the end of the sternum. It seemed to be necessary that the current should be fairly strong, strong enough to produce marked contractions of the upper divisions of the rectus. We found that this caused inspiration alike when the subject was inspiring, as when he was holding his breath. Because of the difficulty of immediately getting the poles into such a position that the phrenics in the neck are faradised, I would recommend this mode of electrical stimulation of the diaphragm. Of the two means, we found that this answered best.

*Accidents which are not especially associated with an Imperfect Anæsthesia.*

C. *Failure of the circulation.*—I. *Causation.*—This may happen from (1) hæmorrhage, (2) reflex inhibition of the heart, and (3) over-dosage. All these conditions are comparatively rare, and they are naturally all more dangerous with chloroform than with ether. Should an asphyxial condition co-exist, the liability of the heart to fail will be much increased.

1. *Hæmorrhage.*—The loss of more than a certain amount of blood will lower the blood-pressure, and so impair the action of the heart, and this particularly when the heart is under the weakening influence of chloroform.

2. *Reflex inhibition of the heart.*—It may seem at first sight strange not to include this under the consideration of those accidents associated with states of imperfect anæsthesia. Certainly when it occurs in those states it is likely to produce a greater effect. The reasons that I think it ought to be considered here are that it has been observed to occur in the deepest anæsthesia consistent with safety; that it is, in practice, most likely to be met with during the performance of the operation, when the anæsthesia will be good; and, lastly, in order to draw a clear distinction between this condition and the shock described on p. 437. The only explanation that one can apply to this event is that it is caused by an efferent stimulus down the vagus from the medulla, that being the only part of the brain that is awake, so to speak,

in deep anæsthesia. The explanation of how the sensory stimulus reaches the medulla is not so clear. Suffice it to say that the stimulus is of a very profound and strong character in these cases. The most common causes of the accident that have been noticed are operations about the kidney, cervix, and rectum; division of the cord in castration; interference with a large nerve, as dividing the optic nerve, or stretching the sciatic; and direct irritation or injury of the vagus. Of this latter causation Dr. F. Hewitt records a most instructive case.[1] He had a patient completely anæsthetised, with flaccidity, abolition of the lid reflex, and stertor. The operation was upon the carotid, and while it was being exposed the face became pale, the pulse extremely feeble, and the respiration shallow. That this was due to interference with the vagus was seen the next day, for it is recorded that when the wound was being examined, and a ligature round the carotid was disturbed, exactly the same symptoms happened. I have been unable to find any record of a death directly due to reflex inhibition of the heart, but the occurrence of dangerous symptoms, with temporary suspension of cardiac action, is by no means rare. Death does not seem likely to happen in such cases unless the general condition known as "shock" is present.

3. *Over-dose.*—It has been seen that the heart may be suddenly paralysed from an over-dose of chloroform when the proportion of chloroform in the inspired air rises above 5 per cent. or thereabouts. An over-dose of chloroform may also be given gradually. The heart, in the case of such an over-dose slowly given, is the cause of death, but the breathing usually ceases first, although, as has been often pointed out, the heart has been actually performing very little work for some time, the character of the pulse being, as was said before, a fallacious guide.

II. *Symptoms.*—The remarks on the symptoms of shock (p. 442) apply also to the symptoms of reflex inhibition of the heart. The symptoms of hæmorrhage and over-dose are distinguished by their more gradual advent. The pulse becomes weaker, and intermits and slows; the pupil gradually dilates; the eyelids are half open; the face

[1] 'Lancet,' 1890, vol. i, p. 515.

gradually assumes the pallid appearance of death, and breathing becomes shallower until it finally fades away. The heart beats in a feeble and utterly ineffective manner for perhaps half a minute after breathing has ceased. It is a cardiac death. Occasionally the breathing may cease very suddenly from an over-dose of chloroform. This is the rule with animals, but it has been shown to be also a purely cardiac death.

III. *Treatment.*—This has already been fully discussed (pp. 443 to 447). It remains to be said here that when, during an operation, good respiration is encouraged the pulse will improve correspondingly. The main point in the treatment of an over-dose of any anæsthetic is to persist with artificial respiration as long as the slightest hope remains.

D. *Impairment of respiration.*—I. *Causation.*—The following causes have to be considered :—(1) Foreign bodies ; (2) asphyxia independently existing ; (3) morbid conditions ; (4) the effect of operations ; (5) failure of breathing from weakened circulation ; (6) the effect of morphia ; and (7) over-dose.

1. *Foreign bodies.*—A large number of cases are recorded in which solid foreign bodies have produced obstruction to breathing of various degrees. When such a substance presses upon, or enters the glottis, alarming symptoms suddenly arise, and death may ensue.[1] A healthy lad, aged 14, was given ether for an operation to unite some divided tendons in a wound of the wrist.[2] The operation lasted half an hour, and was almost concluded when he "suddenly vomited a quantity of pultaceous matter which filled the mouth and fauces," and breathing ceased. He quickly became cyanosed. The mouth was cleaned out, tracheotomy was performed, and artificial respiration was practised. "Spontaneous breathing recommenced, and a quantity of vomited material was forced through the tube." In spite of the continuance of treatment, the breathing again failed, and the patient gradually died one hour and a half after vomiting had occurred. At the autopsy the viscera were healthy, but "wedged into, and quite blocking the right bronchus was a piece of greengage rind rolled up

[1] 'Brit. Med. Journ.,' vol. ii, 1891, p. 21. (Orange peel in larynx.)

[2] Ibid., vol. ii, 1881, p. 414.

into a firm pellet." Many similar deaths have happened. The accidental liquids which may embarrass breathing deserve special notice, as the accident is less preventable, and the causation of the symptoms may not be clear. Blood has thus caused death. A man who had sustained a fracture of his lower jaw died during the administration of chloroform with symptoms of obstructed breathing which could not be remedied. At the necropsy it was found that the larynx had been injured, and there was blood in the trachea.[1] Pus may also give trouble. Mucus is prone to give rise to symptoms of obstruction, more especially when ether is the anæsthetic used. The following case, which Mr. Burghard tells me he witnessed recently, is most instructive. A middle-aged man, suffering from a chronic lung disease in which large quantities of very thick tenacious mucus were secreted, was put under the A. C. E. mixture for a slight rectal operation. The breathing was bubbling from the presence of mucus throughout, but the patient's colour was able to be kept good by constant care. After the operation was completed, and after the anæsthetic had been discontinued for some three minutes and the patient was beginning to show signs of returning consciousness, a violent act of retching took place, during which time the contents of the rectum were expelled. No actual vomiting took place either then or afterwards. This effort of retching, which lasted but a moment, was followed by complete respiratory obstruction. The patient very rapidly became almost black in the face, the jaws were tightly clenched, the pupils widely dilated, and before the mouth could be opened the pulse was very feeble. The mouth was opened with some difficulty, and the tongue forcibly drawn forwards, when, fortunately, it was found, by compression of the chest, that the air-way was patent. No effort of breathing was made by the patient for some time, but artificial respiration soon remedied the condition, and he made a rapid recovery. The obstruction was no doubt due to mucus.

2. *The independent existence of asphyxia.*—An excessive amount of air-limitation, from accident or from design, will conduce to the production of asphyxial symptoms.

[1] 'Lancet,' 1882, vol. ii, p. 540.

3. *Morbid conditions.*—As was pointed out by Dr. F. Hewitt,[1] patients in whom the assistance of extraordinary muscles of respiration is necessary for the maintenance of breathing are particularly prone to exhibit embarrassment of respiration during anæsthesia. These extra muscles are prevented from acting in an early stage of anæsthesia, and much care and skill is required to guide such a patient through a successful administration. Growths, especially enlarged thyroids, and aneurisms are often the cause of much trouble. The fact has been observed that those cases are the most unfavorable in which the respiratory embarrassment is of recent origin, as one would naturally expect from the absence of compensatory conditions.

4. *The effect of operations.*—Any operative interference with any part of the mechanism of respiration will naturally have its effect in impairing breathing. Operations wounding the pleura may be especially mentioned in this connection.

5. *Failure of breathing from weakened circulation.*—It has been seen that breathing fails from this cause, however produced. The functions of circulation and respiration are so connected together that difficulty often arises as to which is the primary factor in producing the accident. Witness the long discussion upon the action of chloroform, from the Hyderabad Commission to the present time.

6. *The effect of morphia.*—I wish more particularly to refer to this as a cause of respiratory failure, because in the latest publication of the New Sydenham Society, vol. i of a translation of Pozzi on Gynæcology, the practice of "mixed anæsthesia" (administering morphia subcutaneously before an anæsthetic) is strongly and ably advocated. On the Continent it has many adherents, but in England the practice is generally looked upon as distinctly predisposing to failure of respiration. Sir J. Lister wrote of the depressing effects of this method in 1882. Dr. Jacob[2] reported three apparently clear cases of deaths referable to this cause. Dr. F. Hewitt has reported a case.[3] Dr. Jacob

[1] 'Med.-Chir. Trans.,' vol. lxxiv, p. 110.
[2] Jacob, 'Brit. Med. Journ.,' 1881, vol. i, p. 69
[3] Hewitt, 'Practitioner,' 1887, p. 103.

draws attention to the fact that five out of fourteen cases of death under ether, with which he was acquainted during ten years, were during operations for obstruction of the bowel, where presumably morphia or opium had been given. In such a case the breathing would seem to gradually become less deep, until it eventually ceases from paralysis of the respiratory centre. Mr. Lucas[1] also expressed the same views in 1882, alluding to two recent deaths during operations for hernia, when morphia had been given. And many other acute observers have spoken to the same effect. The facts appear to be that the previous exhibition of morphia renders less of the anæsthetic necessary, and gives a remarkably quiet anæsthesia; but the depressing effect of the drug upon the respiratory centre, when combined with chloroform or ether in the system, often produces very grave impairment of the act of respiration, and has been responsible for many deaths. In England the method is practically only employed in brain surgery, where it has the advocacy of Professor Horsley; and even then I venture to submit that we have enough evidence to lead us to give the smallest dose of morphia consistent with the production of a quiet anæsthesia. One sixth or one fourth of a grain seems to be ample for this purpose in any case.

7. *Over-dose.*—The failure of respiration during an over-dose of chloroform has recently been clearly shown by Drs. Gaskell and Shore[2] to be mainly cardiac in origin. No doubt if the heart held out direct paralysis of the respiratory centre would occur, but the heart is brought to a standstill by the poisonous action of the chloroform before this happens, and before the heart actually stops the breathing usually fails, often suddenly. Ether, in large and prolonged doses, has been known to cause death explicable in no other way but that of direct respiratory paralysis. Nitrous oxide produces obstruction to breathing when pressed to an over-dose, in nearly every instance. A remarkable instance of direct respiratory paralysis, without obstruction, and from what must be regarded as unique idiosyncrasy, occurred to me lately. As I have said little about idiosyn-

[1] Lucas, 'Brit. Med. Journ.,' 1882, vol. i, p. 500.

[2] Gaskell and Shore, loc. cit.

crasy, and as this case furnishes an excellent example, I propose to report it here. The patient was a medium-sized, well-nourished, florid girl, aged 23, and by no means nervous. She was to have nitrous oxide for the extraction of two teeth. She could not be induced to breathe deeply. After only ten short breaths a very shallow one was taken in the same rhythm, and breathing absolutely ceased. The lid-reflex was absent: pupils small: globes fixed. The face-piece was kept on for fifteen seconds after breathing ceased, when it was removed, and the operator was requested to proceed. While the two teeth were being extracted I pressed upon the chest once, and air escaped with great ease. Her colour was quite good until the first tooth was removed, from which time it gradually deepened. There was no obstruction to breathing, as I verified by seizing the tongue and drawing it forward as soon as the second tooth was removed, and passing my finger to the back of the throat. She was absolutely quiet and relaxed throughout, and no effort at inspiration was apparent after the breathing ceased. Considering the extraordinary sequence of events, I examined as to whether she was tight-laced, intended to rip open the stays, but I found that her dress and stays were quite open. No reflex occurred when the finger was passed to the back of the throat. The pulse was good throughout. As by this time she was deeply cyanosed, the patient was placed upon the floor and artificial respiration was performed. Air entered and left the chest with the greatest ease, and her colour rapidly became normal, but it was quite half a minute before she breathed, and a few seconds afterwards she was quite well. Not being clear as to the explanation of the case, and having a few days later to narcotise her again, I determined to give her gas and air. She breathed no more deeply than before. The first seven inspirations were of gas, the eighth inspiration was air, the next four were gas, and the thirteenth and last, which was very shallow, was air. She then behaved in every respect as on the first occasion. Two teeth were removed. Having satisfied ourselves that no obstruction was present, we watched her, as before, perfectly quiet and relaxed and with a full good pulse, grow

gradually livid. This time the chest was compressed while she was in the chair, and before she became so deeply cyanosed as on the first occasion. After about ten compressions she breathed, and came round naturally and rapidly. She had not been taking morphia, and the only fact I could elicit about her which was abnormal was that she suffered from "rheumatism in the shoulders."

II. *Symptoms.*—What was said on p. 451 applies to most of the foregoing conditions. In the case of an over-dose, and when morphia has been given, the muscles are usually flaccid. A considerable proportion of deaths under ether have occurred in operations for hernia and intestinal obstruction when morphia was present in the system. As the symptoms have been those of depression, some of these deaths have been reported as due to shock (p. 436). In dealing with such a case the action of morphia in anæsthesia should be remembered. There is one point more which deserves special notice. It was well put by the late Dr. Sheppard,[1] who said that the differences between the shallow breathing of recovery and that of dangerously deep anæsthesia were most difficult to appreciate, and that this constituted one of the chief difficulties in the administration of chloroform. Other signs must be consulted and relied upon when in doubt on this point. Of these signs the size of the pupil is one of the most important. With an over-dose of any anæsthetic it gradually dilates.

III. *Treatment.*—This has also been mostly described on p. 452. For minor degrees of impaired breathing, the lips and cheeks may be rubbed with a rough towel (Hewitt). The treatment in a case of vomiting should be to place the head on one side, keep the mouth slightly open (by means of an india-rubber prop if necessary) and the lower jaw well forward. Strychnine was found by Dr. Wood[2] to have a great and immediate effect in stimulating the breathing. Five minims of the liquor may be injected when indicated by feebleness of the respiratory efforts. Artificial respiration, by Silvester's method, is the most important

[1] Sheppard, 'Brit. Med. Journ.,' 1890, vol. ii, p. 1366.

[2] Wood, op. cit.

means of treating all forms of cessation of respiration, and it should be continued as long as the least hope remains. Foreign bodies should be removed, with finger or forceps, as soon as evidence of their presence is apparent. Liquids can be prevented from doing harm by the position of the head, and may be mopped out with a sponge from time to time. When a foreign body gets into the larynx, inversion and slapping the back may be rapidly tried. No time, however, should be lost in opening the trachea when milder measures fail, and often the offending substance may be sucked out. After artificial respiration, performed for a considerable time, has been recovered from, the patient must be carefully watched lest the so-called "secondary apnœa"[1] of Linhart should occur. For some time after a patient has recovered from impending death by asphyxia, he appears to be liable to failure of respiration, which may cause death.

*Remarks upon the modes of action of ether and chloroform.*—Various allusions to this subject have been made in the foregoing pages, but it may be useful to set down here the main facts concerning the action of these agents upon the vital organs. Chloroform at once stimulates the vaso-motor and other centres in the medulla, and directly impairs the muscle of the heart. After a time, the poisonous effects upon the heart still proceeding, it depresses the centres referred to, including the respiratory centre. Deaths from over-doses of chloroform are brought about by its effect upon the heart. Ether raises the blood-pressure and increases the force of the heart-beat. It eventually paralyses the respiratory centre. In an over-dose ether thus kills from asphyxia. Occasionally, if a great lack of oxygen exists, the heart may suddenly fail. All mixtures containing chloroform (A. C. E., "methylene," chloroform and alcohol, &c.) are to be looked upon as diluted chloroform. As regards nitrous oxide, it may be said that practically the only dangers that occur in narcosis produced by it are those of several forms of respiratory obstruction, which pass off very rapidly. Superadded syncope is most rare. Even with nitrous oxide and oxygen I have met with one case

[1] Harley, 'System of Surgery,' vol. iii, p. 830.

that might have been troublesome. A patient with a full stomach vomited into the face-piece during the inhalation.

*Summary.*

The accidents of anæsthesia have been seen to be more dangerous to life when chloroform is the agent employed, because of the element of heart-failure thereby introduced. Ether possesses the great advantage over chloroform that in states of danger the patient can so much better resist the conditions threatening life. Accidents occur most frequently in states of imperfect anæsthesia, and they are here especially dangerous in chloroform narcosis. Deaths from ether are most rare in the healthy.[1]

The following are some rational points of practice. Chloroform should be given freely and continuously until the patient is completely anæsthetised. It is best given by the open method, as with a Junker's inhaler a misplaced reliance may be felt in the apparatus. With a drop-bottle one always approximately knows the amount one is giving. Having once piloted the patient into the calmer and safer waters of good anæsthesia, it should be the aim of the administrator to keep him there. Sir J. Lister's observation,[2] that it is necessary to use the drop-bottle about half as frequently to keep up anæsthesia as to induce it, will be found a useful guide in practice. Should any doubt exist in the mind of the anæsthetist as to the degree of anæsthesia present, as I have often felt, the chloroform should be withheld until the conjunctival reflex just reappears. When chloroform is properly given the risks of heart-failure, from any cause, can be almost abolished. Surgeon-Lieutenant-Colonel Lawrie's large number of uniformly successful administrations prove this.

Ether should be given upon the same general principles of rapidly and safely passing through the state of incomplete narcosis. A due amount of oxygen should be allowed, whatever apparatus is used.

In giving any anæsthetic the first and constant attention

[1] Jacob, 'Brit. Med. Journ.,' 1885, vol. i, p. 887.

[2] Lister, op. cit., p. 619.

of the anæsthetist should be directed to the breathing. At every moment of the time during which the patient is in his hands he should be cognizant of the character of the last breath taken, which is simply a matter of practice. As we have seen, he should also keep himself informed of the state of the pulse, but never at the expense of neglecting the former duty. The accidents in which the symptom of pulse-failure is the chief feature are to be prevented from happening, rather than sought for to be cured.

As Mr. Holmes has observed, "experience and care can reduce the risk of death under chloroform as well as ether to a very insignificant danger."

# APPENDIX TO THE CATALOGUE

OF THE

# PATHOLOGICAL MUSEUM OF GUY'S HOSPITAL,

# 1892.

---

By LAURISTON E. SHAW.

---

**1817**(50) **Pouch in the Duodenum.**

The pyloric end of a stomach, with a portion of the duodenum laid open to show a globular pouch, about an inch in diameter, projecting from the outer wall of the intestine one inch from the biliary papilla. The wall of the pouch is thin and free from adhesions. There is a healed gastric ulcer situated upon the lesser curvature an inch from the pyloric ring.

Mary W., æt. 72, was admitted under Dr. Hale White for chronic Bright's disease, and died from uræmia. *See Insp.*, 1892, No. 13.

**1817**(52) **Pouch in the Duodenum.**

A portion of a duodenum shewing a globular pouch, the mouth of which measures about an inch in diameter, situated by the side of the papilla at the opening of the pancreatic and common bile-ducts. The mucous membrane lining the sac is normal, and its wall is stretched and thin. The pouch is not adherent to the neighouring structures.

Jane B., æt. 74, was admitted under Mr. Durham for a strangulated femoral hernia. Herniotomy was performed, and she died three days afterwards. *See Insp.* 1889, No. 356.

**1817**(53) **Papilloma of the Duodenum.**

A portion of a duodenum mounted to shew a cauliflower-like mass of growth having a broad attachment to the mucous membrane around and below the papilla of the common bile-duct, into which a yellow rod has been inserted. The mass is about one and a half inches long, three quarters of an inch broad, and projects half an inch above the surrounding mucous membrane. Close to the growth at its upper part there is a small shallow pouch in the wall of the duodenum.

Elizabeth D., æt. 49, was admitted under Dr. Goodhart for jaundice with pyrexia and hæmorrhage from the intestine. She died three months from the commencement of her illness. At the autopsy two chronic ulcers were found in the rectum, and there were numerous small abscesses in the liver. The common bile-duct was not obstructed. *See Insp.* 1890, No. 385.

**1817**(80) **Laceration of the Duodenum.**

The upper half of a duodenum with a portion of the stomach. Two inches from the pylorus there is, in the duodenum, an irregular perforation which in the recent state admitted the middle finger. On the reverse of the specimen the mucous membrane is seen to project through the opening; the peritoneal coat is lacerated and partly detached.

Emily H., æt. 14, was admitted under Mr. Lucas in a state of collapse, having been struck on the abdomen by a swing. She died twelve hours after admission. At the autopsy there was general peritonitis. *See Insp.* 1889, No. 257.

**1817**(81) **Perforation of the Duodenum.**

The first inch of a duodenum, with the adjoining portion of the stomach mounted to illustrate perforation from without. The peritoneal surface of the duodenum exhibits on its anterior aspect an oval ulcer three quarters of an inch in its longest diameter, situated just beyond the pylorus.

The edges of the ulcer are clean-cut, and in its base, which is formed by mucous membrane, is seen a small perforation, through which, in the recent state, pus escaped into the intestine. Elsewhere the outer surface of the stomach and duodenum is eroded by superficial ulceration and roughened by a deposit of fibrinous material.

Emma L., æt. 18, was admitted under Dr. Goodhart with signs of chronic peritonitis. She died eight months after the commencement of her illness. At the autopsy the uterus and Fallopian tubes were found to be tuberculous; the abdominal cavity was distended with gas, and there were numerous collections of pus between the matted intestines. *See Insp.* 1889, No. 327.

**1817**(82) **Pancreatico-duodenal Fistula.**

A duodenum with the pancreas and a part of the stomach mounted to shew a communication between the duodenum and a cavity in the head of the pancreas, which in the recent state contained blood-clot. The perforation is situated on the concave border of the duodenum two inches below the pyloric ring, and an inch and a half above the biliary papilla. Histological examination of the walls of the cavity in the pancreas shews an excess of fibrous tissue between the acini of the gland, but no evidence of malignant growth.

Lizzie C., æt. 29, was admitted under Dr. Hale White for severe hæmatemesis and melæna, which began eight days before admission. Five days later an exploratory laparotomy was performed, but the source of hæmorrhage was not discovered. She died on the following day. *See Insp.* 1891, No. 357.

**1819**(20) **Diverticulum of the Ileum.**

A portion of small intestine with a diverticulum projecting from its lateral wall. The pouch is an inch in length, half an inch in diameter, and presents two short lateral projections from its blunt extremity.

James R., æt. 31, was admitted under Dr. Newton Pitt for a swelling in the right loin, and died from sarcoma of the supra-renal capsule. *See Insp.* 1889, No. 376.

**1830**(6) **Perforating Ulcer of the Duodenum.**

A pylorus with the adjacent parts of the stomach and duodenum laid open to shew a perforating ulcer of the intestine. The perforation is oval, measuring half an inch in its longer diameter, and is situated upon the anterior wall half an inch from the pylorus. Its edges are abrupt, and the intestinal wall around it is somewhat thickened. Immediately opposite to it, upon the posterior wall, there is a second ulcer, similar in shape and size, whose base is formed by the thickened serous coat of the bowel.

Frederick G., æt. 21, was admitted under Dr. Perry eighteen hours after the onset of an acute attack of abdominal pain, shortly followed by vomiting and collapse. Laparotomy was performed, and a large quantity of dark brown acid fluid, without fæcal odour, was evacuated from the peritoneal cavity; there was much lymph upon the coils of intestine. Four hours later the patient died. *See Insp.*, 1892, No. 238.

**1831**(18) **Enteritis.**

The last foot of an ileum laid open to shew two inflamed areas, the lower one immediately above the ileo-cæcal valve, and the other five inches higher up. At the inflamed parts the intestine is thickened and contracted, and its mucous membrane exhibits transverse folds, which are covered by loosely adherent granular lymph. Histologically all the coats of the bowel are seen to be infiltrated with small round cells.

Mary S., æt. 35, was admitted under Dr. Newton Pitt for hæmatemesis with severe anæmia. She became comatose, and died five days after her admission. At the autopsy the left lobe of the cerebellum was found to be softened, and there was a large chronic ulcer in the stomach. There were a few patches of superficial ulceration in the upper part of the colon. *See Insp.*, 1891, No. 292.

**1844**(56) **Tuberculous Strictures of the Ileum.**

A portion of an ileum laid open to shew two strictures resulting from contraction following tuberculous ulceration. At the seat of constriction the gut is narrowed to about one half its normal diameter, and its lumen is crossed by several fibrous bands covered by mucous membrane. Histologically

the muscular and submucous coats are infiltrated by small cells, some of which have undergone caseation. There are a few giant-cells.

Sarah L., æt. 18, was admitted under Dr. Goodhart for abdominal pain and diarrhœa of about one year's duration. She died the day after her admission, and at the autopsy the intestines were firmly matted together, and the pelvis contained a pint of foul sero-pus. There were several chronic ulcers scattered through the ileum, and there was a perforation in the cæcum. There was no evidence of tuberculous disease in any other part of the body. *See Insp.*, 1891, No. 24.

**1844**(61) **Tuberculosis of the Ileum.**

Two portions of the lower end of an ileum shewing a general enlargement of the agminated and solitary glands, which histologically are seen to be infiltrated with characteristic tuberculous deposit. There is little if any ulceration.

Alfred W., æt. 23, was admitted under Dr. Pye-Smith with pyrexia and the physical signs of tuberculosis of the lungs. He had been ill for three years, and died a few days after his admission. At the autopsy there was old and recent tuberculous disease in the lungs, with dilatation of the bronchial tubes. *See Insp.*, 1891, No. 417.

**1845**(54) **Sarcoma of the Ileum.**

Three portions of an ileum, each of which shews a discoid mass of growth projecting about a quarter of an inch above the mucous membrane, the largest measuring two and a half inches, and the smallest about one inch in diameter. In all the specimens the growth presents an ulcerated surface, and in one it has penetrated the wall of the intestine, which has yielded, producing a globular pouch projecting externally. In the wall of the pouch there is a perforation, which in the recent state was closed by adhesion to a neighbouring coil of intestine. Histologically the growth is a round and oval-celled sarcoma.

William L., æt. 45, was admitted under Mr. Jacobson for a growth in the right tonsil, which was first noticed seven weeks before admission. He died about six weeks later, and at the autopsy secondary deposits were found in the cervical and bronchial glands and in the pleura. *See Insp.*, 1891, No. 343.

**1845**(56) **Multiple Carcinomatous Contractions of the Ileum.**

A piece of the lower end of an ileum laid open to shew three annular constrictions produced by the contraction of secondary malignant deposit in the serous coat. The mucous membrane at the seat of stricture is free from ulceration. Histologically the growth is a spheroidal-celled carcinoma.

Jemima H., æt. 38, was admitted under Dr. Shaw for intestinal obstruction of ten days' duration. Laparotomy was performed, and the patient died the following day. At the autopsy the right ovary was enlarged by a deposit of spheroidal carcinoma. There was a similar growth in the wall of the stomach which was thought to be secondary. *See Insp.*, 1891, No. 191.

**1854**(54) **Carcinomatous Ulcer of the Colon.**

A portion of a colon shewing an oval transverse ulcer, which was situated three feet from the ileo-cæcal valve. The ulcer measures an inch in its longest diameter, and its base is formed by thickened submucous tissue. Its border is considerably raised above the surrounding mucous membrane, and on histological examination is found to be infiltrated by a growth having the characters of a cylindrical-celled carcinoma. About four inches above the ulcer is seen a button-like polypus half an inch in diameter, the mucous membrane of which is somewhat blackened from congestion.

Matilda M., æt. 69, was admitted under Dr. Perry for acute peritonitis, of which she died twelve hours later. At the autopsy the peritonitis was found to be caused by suppuration of the gall-bladder. *See Insp.*, 1892, No. 272; and *Prep.*, 1873 (10).

**1854**(56) **Carcinoma of the Colon opening the Stomach.**

The transverse arch and splenic flexure of a colon, with the spleen and stomach. Encircling the colon, just above the splenic flexure, there is a mass of growth, two inches wide, the surface of which is deeply ulcerated. In the transverse colon at the border of the growth there is a perforation, indicated by a red rod, communicating with the stomach at the middle of its posterior wall an inch from the greater curvature. Blue rods have been passed from the bowel into

two sinuses, which in the recent state communicated with abscess cavities between the neighbouring viscera. Above the growth the transverse colon is greatly dilated, measuring six inches in circumference. Histologically the growth is a cylindrical-celled carcinoma.

John S., æt. 36, was admitted under Dr. Goodhart for abdominal pain of four months' duration. Vomiting came on three weeks before admission, and four days afterwards the material vomited was distinctly fæcal. An exploratory laparotomy revealed a large mass of growth in the left hypochondriac region. Right inguinal colotomy was performed, and subsequently an artificial anus was made in the small intestine. The patient died eight days after admission. At the autopsy the small intestine was found to have been opened eighteen inches below the duodenum. There were no secondary deposits of growth. *See Insp.*, 1892, No. 231.

**1857**(16) **Tuberculous Ulceration of the Colon.**

A portion of a colon, the mucous membrane of which is almost entirely destroyed by ulceration. The ulcerated surface is roughened, and in some places the muscular coat is exposed. The edges of the persistent mucous membrane are irregular in outline, prominent, and undermined.

Philip W., æt. 25, was admitted under Dr. Pavy for wasting and constant diarrhœa of three months' duration. He died ten days after admission, and at the autopsy there was tuberculous disease of the lungs and larynx. There were numerous ulcers in the small intestine, in which the bacilli of tuberculosis were found. *See Insp.*, 1890, No. 86A.

**1857**(17) **Tuberculous Nodule in the Colon.**

A portion of a colon shewing an oval nodule measuring about an inch in length, and projecting a third of an inch from the mucous membrane. There is a small patch of superficial ulceration at the central part of the nodule. Histological examination shews that the submucous tissue is infiltrated with tuberculous deposit.

Mary Y., æt. 42, was admitted under Dr. Pye-Smith for phthisis and tuberculous laryngitis, from which she died. At the autopsy a few tuberculous ulcers were found in the ileum. *See Insp.*, 1890, No. 314.

**1859**(36) **Ulcerative Colitis.**

An anus with the last ten inches of the large intestine shewing two ulcerated areas, one just within the anus, and

the other at the upper part of the preparation. The intervening mucous membrane appears to be healthy.

William H., æt. 54, was admitted under Mr. Jacobson for a stone in the bladder, which was removed by supra-pubic lithotomy. Two months after the operation troublesome diarrhœa supervened, and he died ten days later. At the autopsy the whole of the large intestine was ulcerated, the ulceration in parts being limited to the solitary follicles. *See Insp.*, 1890, No. 379.

**1860(5) Ulcerative Colitis.**

A portion of a colon, the mucous membrane of which is in parts completely destroyed by ulceration, and in parts separated from the deeper tissues. The exposed muscular coat of the bowel is crossed by bridles of mucous membrane, beneath which rods have been passed. In the regions where the destructive process is less advanced the ulcers are small, round, and punched-out, and their edges are thin and undermined.

Sarah W., æt. 25, was admitted under Dr. Goodhart for high fever, with diarrhœa and abdominal pain of three days' duration. She died three weeks later, having passed blood in her motions for the first time the day before her death. At the autopsy the small intestine was normal, and the condition shewn in the preparation was found throughout the colon. *See Insp.*, 1890, No. 309.

**1864 Ulceration of the Colon in Pneumonia.**

Portions of a colon, mounted to shew numerous irregular shallow ulcers, which for the most part have a transverse direction. In the bases of some of them the muscular coat is exposed.

Henry C., æt. 32, was admitted under Dr. Pye-Smith for acute pneumonia, complicated with delirium tremens of five days' duration. On the eleventh day of his illness diarrhœa supervened, and six days later he died. At the autopsy the lobar pneumonia was in a condition of resolution, but there were numerous patches of more recent lobular inflammation. The small intestine was healthy. *See Insp.*, 1890, No. 382.

**1864(3) Ulcers of the Colon.**

A portion of a colon, laid open to shew numerous round ulcers, varying from an eighth to three quarters of an inch

in diameter. In some of them the muscular coat of the intestine is exposed, in others the floor is covered with a thin pellicle.

Joseph L., æt. 51, was admitted under Mr. Lane for a fracture of the spine in the lumbar region with complete paraplegia. The spine was trephined, and the patient died fourteen days after the accident, having suffered for some days from profuse diarrhœa. At the autopsy a similar condition to that shewn in the preparation was found throughout the large intestine. *See Insp.*, 1890, No. 283.

**1873**(10) **Polypus of the Colon.**

A portion of the sigmoid flexure of a colon, shewing a pedunculated polypus attached to its mucous membrane by a stalk about an inch and a half in length. The polypus is oval in shape, measuring about an inch in length and five eighths of an inch across; the mucous membrane covering it is traversed by irregular fissures. Histologically it consists of a central core of fibrous and muscular tissue, covered with branched villous processes of mucous membrane.

Matilda M., æt. 69, was admitted under Dr. Perry for acute peritonitis, of which, twelve hours later, she died. At the autopsy the peritonitis was found to be caused by suppuration of the gall-bladder. *See Insp.*, 1892, No. 272, and *Preps.*, 1854 (54).

**1876**(60) **Membranous Colitis.**

A portion of a colon, shewing upon its mucous membrane linear incrustations of granular lymph, corresponding in position with the longitudinal muscular bands of the large intestine. Histologically the mucous membrane is seen to be infiltrated with small round cells, and there is some thickening of the submucous tissue.

William W., æt. 39, was admitted under Mr. Davies-Colley for cellulitis of the neck of six days' duration. An abscess containing foul pus was opened, and the patient died one week after admission. At the autopsy there was necrosis of the thyroid cartilage, and the mucous membrane of the first part of the colon was congested. *See Insp.*, 1891, No. 118.

**1877**(10) **Membranous Colitis from Injury.**

A portion of an ascending colon, laid open to shew a thin deposit of granular lymph upon its mucous membrane,

which in the recent state was deeply injected. On the reverse of the specimen the muscular and serous coats are seen to be lacerated in several places, and there is adherent decolourised blood-clot.

Thomas M., æt. 4, was admitted under Mr. Howse, having been run over by a cab. The front wheel passed over and fractured his right thigh, and the hind wheel crossed his abdomen. He died on the third day from the accident, and at the autopsy there was hæmorrhage into the cellular tissue of the right iliac fossa and into the peritoneum. There was no peritonitis. *See Insp.*, 1891, No. 108.

**1880(16) Stump of Cæcal Appendix.**

A cæcum mounted to shew the condition of the parts one month after amputation of the vermiform appendix. There is complete union.

Margaret H., æt. 16, was admitted under Dr. Goodhart with symptoms of perityphlitis of eight days' duration. A fortnight after admission the vermiform appendix was removed by Mr. Lane. She died a month later from pyæmia. *See Insp.* 1890, No. 149.

**1880(51) Ulceration of Cæcal Appendix and Cæcum.**

A portion of a colon with the cæcum and its appendix. At its origin the appendix presents a considerable dilatation, from which a concretion has been removed. The mucous membrane of the appendix at this part and of the cæcum is extensively ulcerated and undermined. There is another patch of superficial ulceration at the tip of the appendix, where a second concretion was lodged.

Ellen P., æt. 14, was admitted under Dr. Hale White for a cerebellar tumour, from which she died suddenly. There were no symptoms of intestinal disease. *See Insp.* 1889, No. 412.

**1881(5) Sloughing Cæcal Appendix.**

A cæcum and the vermiform appendix, with a portion of the colon. The appendix which is adherent to the back of the colon is about four inches long, and is perforated in three places. Through the large oval opening near its cæcal attachment a fæcal concretion was removed. There is recent lymph upon the serous covering of the intestine.

George L., æt. 18, was admitted under Dr. Pavy for vomiting and pain in the right side of the abdomen of two days' duration. On admission there was a hard tender lump in the right iliac region. He died on the fifth day of his illness, with symptoms of general peritonitis. At the autopsy the intestines were found to be matted together with recent lymph. *See Insp.* 1889, No. 254.

**1881(81) Lymphadenoma of the Cæcal Appendix.**

A vermiform appendix with the cæcum and a portion of the ileum, shewing a general overgrowth of lymphoid tissue. The mucous membrane of the appendix is markedly rugose, and much thickened by a deposit of small round-cells in the submucous tissue. Elsewhere the agminated and solitary glands are unusually prominent. The lymphatic glands are also enlarged.

Harry B., æt. 15, was admitted under Dr. Pye-Smith for Hodgkin's disease, and died seven months from the onset of his illness. At the autopsy there was enlargement of the lymphatic glands, liver, and spleen. *See Insp.* 1891, No. 344.

**3011a Foreign body removed by Laryngotomy.**

Two portions of cancellous bone, the larger measuring three quarters of an inch in its longest axis, which were firmly impacted below the vocal cords, and were removed by the operation of laryngotomy.

William C., æt. 63, was admitted under Dr. Taylor in 1881 for cough and expectoration with very slight laryngeal stridor. The laryngoscope showed a fragment of bone fixed within the larynx. Mr. Golding-Bird performed laryngotomy and extracted the foreign body. The bone stuck in the man's throat four months before the operation, and was forced into the larynx by his wife while endeavouring to dislodge it with a table-spoon. He was discharged well a fortnight after its removal. *See Clin. Soc. Trans.*, vol. xvii, p. 214.

**3020a Membranous Laryngitis following Measles.**

A larynx and upper part of the trachea, the mucous membrane of which is covered by a thin layer of adherent false membrane.

Ethel C., æt. 2, was admitted under Dr. Hale White for laryngeal obstruction, the symptoms of which were first noticed five days after the

appearance of the rash of measles. She died rather suddenly forty-eight hours after the onset of the obstruction. *See Insp.* 1890, No. 398.

**3160a Tracheo-œsophageal Fistulæ; Epithelioma of the Œsophagus.**

A larynx and trachea with the œsophagus. The trachea has been laid open from the front to shew upon its posterior wall halfway between its bifurcation and the cricoid cartilage two rounded openings with raised warty edges. These openings lead to a small cavity behind the trachea which also communicates with the œsophagus by means of two oval perforations. At the junction of the œsophagus and pharynx there is an irregular ulcer, the edge of which is seen under the microscope to be infiltrated with a growth of squamous-celled epithelioma.

William J., æt. 57, was admitted under Mr. Howse for difficulty in swallowing of one month's duration, and died on the following day from gangrenous broncho-pneumonia. *See Insp.* 1891, No. 316.

**3258a Compressed Lung.**

A right lung, the upper lobe of which is somewhat distended and covered with flocculent adhesions. The middle and lower lobes are much shrunken and almost solid; the pleura covering them is thickened and for the most part quite smooth.

Ada V., æt. 24, was admitted under Dr. Newton Pitt for chronic endocarditis, with the signs of effusion of fluid into the right chest. It was stated that pleuritic effusion had existed for the preceding eleven months. At the autopsy thirty ounces of serum were found in the right chest, and the aortic and tricuspid orifices were narrowed. *See Insp.* 1891, No. 419.

**3283a Gangrenous Pneumonia; Intra-pleural Hæmorrhage.**

A right lung, a portion of the upper lobe of which has been removed to exhibit, towards its base, a ragged cavity about an inch in diameter. The lung tissue around the cavity is consolidated by pneumonia. Within the cavity the blue rod indicates a large branch of the pulmonary artery,

the ulceration of which led to fatal hæmorrhage. A communication between the excavation in the lung and the pleural cavity is marked by a red rod. There is recent lymph upon the pleura.

William L., æt. 45, was admitted under Dr. Perry for hæmoptysis and pyrexia, with dulness at the lower part of the front of the right chest. He died the day after admission with symptoms of internal hæmorrhage. At the autopsy 52 ounces of clotted blood were found in the right pleural cavity. *See Insp.* 1891, No. 119.

**A3323 Basal Phthisis.**

A section through a right lung, the lower lobe of which is contracted and presents a considerable excess of fibrous tissue. The bronchial tubes in this lobe are somewhat dilated, and the pulmonary tissue is in parts consolidated by caseous broncho-pneumonia. There are one or two small ragged excavations in the lung. The upper lobe is unaffected. There is considerable thickening of the pleura, more marked at the base than at the apex.

Walter S., æt. 21, was admitted under Dr. Goodhart with a very deformed chest and symptoms of chronic lung disease. He had had a bad cough for many years. There were physical signs of consolidation of the lower part of both lungs, and tubercle bacilli were found in the sputum. At the autopsy the lower lobes of both lungs were found to be similarly affected, and there was a patch of tuberculous consolidation at the anterior part of the left upper lobe. The right side of the heart was dilated. A caseous nodule was found in one of the kidneys. *See Insp.* 1890, No. 471.

**3428a Secondary Sarcoma of the Pleura.**

A portion of parietal pleura, projecting from the serous surface of which are numerous secondary deposits, most of which are about the size of a split pea. At the upper part of the preparation there is a large ovoid mass of secondary growth measuring four and a half inches in its longest diameter. Histologically the growth is a very vascular spindle-celled sarcoma.

Anne G., æt. 59, was admitted under Mr. Lucas for a periosteal sarcoma of the lower end of the femur, which was first noticed six

months previously. The limb was amputated through the middle of the thigh, and the patient died six weeks later with symptoms of secondary growth in the thorax. At the autopsy secondary deposits were found in the lung and upon the pericardium. *See Insp.* 1891, No. 306.

**A3464 Enchondroma of the Submaxillary Gland.**

One half of an enlarged submaxillary salivary gland measuring two and a half inches in length and two inches in breadth. Its surface is nodular, and the cut section presents numerous semi-transparent areas. Histologically the gland tissue is infiltrated by a growth of cartilage.

From a man aged about 40.
Presented by Mr. ARBUTHNOT LANE, 1891.

**A3499 Hemiatrophy of the Tongue.**

A tongue with the neighbouring parts, mounted to shew the disproportion in size between the two lateral halves of the organ. The partial atrophy of the left side, which was more obvious in the recent state, is due to the pressure exerted upon the hypoglossal nerve by the mass of carcinomatous glands on the left side of the larynx. Histological examination shews that there is a deposit of squamous-celled epithelioma in the cervical glands, and that the affected nerve and muscles have undergone degeneration.

Matthew J., æt. 60, was admitted under Mr. Durham for a tumour of the left side of the neck and paralysis of the corresponding half of the tongue. The swelling had been noticed for seven weeks. A portion of the tumour was removed, and the patient died two months after admission. The primary seat of growth was not discovered. *See Insp.* 1890, No. 483.

**A3534 Tuberculous Ulceration of the Pharynx.**

A larynx with the pharynx and upper part of the œsophagus. There is severe ulceration partially destroying the epiglottis and the arytæno-epiglottidean folds, and extending to the anterior and lateral walls of the pharynx.

Hannah B., æt. 22, was admitted under Dr. Perry for symptoms of phthisis, with dyspnœa and pain in swallowing. She was seven months pregnant, and died a week after her premature confinement. At the autopsy extensive tuberculous excavation of the lungs was found, and there were numerous ulcers in the ileum. *See Insp.* 1891, No. 157.

**3547a Epithelioma of the Pharynx; Œsophagostomy.**

A larynx and trachea with the pharynx and œsophagus, shewing the gullet narrowed and encircled by an ulcerated growth which extends from the interarytænoid space downwards for about two inches. On the left lateral wall of the œsophagus, half an inch below the inferior limit of the growth, there is a rounded opening which communicated with the front of the neck by a wide channel between the trachea and the common carotid artery. Histologically the growth is a squamous-celled epithelioma.

Eliza G., æt. 60, was admitted under Mr. Davies-Colley for a stricture of the pharynx through which no instrument could be passed. There had been dysphagia for six months before admission. Œsophagostomy was performed, and the patient died six days later. At the autopsy recent lymph was found upon the left pleura, and there were several patches of broncho-pneumonia, one of which was gangrenous. *See Insp.* 1891, No. 204.

**3614a Dilated Hour-glass Stomach; Pyloric Stenosis.**

A greatly dilated stomach divided into two pouches by a constriction situated nearer to the pyloric than to the cardiac end. The ength of the cardiac sac is about eight inches, and of the pyloric nearly twenty, while the transverse diameter of each at the widest part is five inches and a half. In the recent state these pouches contained 40 and 90 fluid ounces respectively. The thumb could be easily passed through the constriction, at which point the gastric wall is somewhat thickened and indurated. Upon the lesser curvature towards the cardiac side of the prominent ridge of mucous membrane surrounding the constriction there is a small oval ulcer with punched-out edges and a smooth base. There is a second similar but somewhat larger ulcer just within the pylorus upon the anterior wall of the stomach. The pyloric ring is narrowed so as barely to admit the little finger.

Jane L., æt. 38, was admitted under Dr. Hale White for constant vomiting, from attacks of which she had suffered for several years. Some relief was afforded by washing out the stomach. She died ten days after

admission from coma following general convulsions. At the autopsy the brain and kidneys were normal. *See Insp.* 1891, No. 301.

**3658 Chronic Gastric Ulcer.**

A stomach laid open to shew an irregular ulcer about two inches in diameter, situated upon the lesser curvature, rather nearer to the pyloric than to the œsophageal opening. The floor of the ulcer is smooth, and in the exposed muscular coat are seen the orifices of several small blood-vessels. Towards the pylorus the edge of the ulcer is flat and undermined, whilst elsewhere the surrounding mucous membrane is hypertrophied, and presents a papillomatous appearance.

From a gentleman, æt. 64, who suffered from gastric symptoms for eighteen months, and died from the effects of hæmorrhage into the stomach.

Presented by Dr. GOODHART, 1889.

**3667 Perforating Gastric Ulcer.**

A portion of a stomach shewing close to the anterior margin of the pyloric orifice a triangular perforation with thick clean-cut edges. Around the perforation the wall of the stomach is considerably thickened, and its serous surface is coated with recent lymph. Histological examination of the border of the ulcer shews no evidence of malignant growth.

Edward W., æt. 51, was admitted under Dr. Newton Pitt for a sudden attack of abdominal pain and collapse, and died twenty-four hours from the onset of his illness. He had suffered from indgestion for twenty-four years, and two years before his death had a profuse attack of hæmatemesis. At the autopsy the peritoneal cavity contained gastric contents, and there was general peritonitis. *See Insp.* 1887, No. 200.

**3685 Hypertrophied Pylorus.**

A stomach, the coats of which are slightly hypertrophied throughout. A section through the pylorus shews a fusiform thickening of this part, about an inch in length, due to a more considerable hypertrophy of its muscular wall. In the recent state a small probe could with difficulty be passed through the pyloric orifice.

Albert E., æt. 5 weeks, was admitted under Dr. Goodhart for vomiting of eight days' duration, with constipation. He died in convulsions thirteen days after admission. At the autopsy the other viscera were normal. *See Insp.* 1891, No. 91; and *Trans. Path. Soc.*, vol. xliii.

**3708 Spheroidal-celled Carcinoma of the Stomach.**

A stomach, laid open along its lesser curvature to shew a rounded area, roughly corresponding to the fundus of the organ, over which the mucous membrane is hypertrophied and markedly rugose, while beneath it the walls of the viscus are thickened and leathery to the touch. Histological examination shews that the submucous and muscular coats are infiltrated by a growth having the character of spheroidal-celled carcinoma.

Presented by Mr. A. T. Davies, 1887.

**3712 Spheroidal-celled Carcinoma of the Stomach (Colloid).**

A stomach laid open to shew a slightly raised ulcerated growth extending from just within the pyloric ring along the lesser curvature for a distance of five inches, and affecting both walls of the organ so as almost to reach the greater curvature. The edges of the growth are raised and slightly everted, and its ulcerated surface presents numerous gelatinous-looking nodules. On the reverse of the specimen the growth is seen to project as a lobulated mass beneath the serous coat. Histologically it is a spheroidal-celled carcinoma, many of the cells of which have undergone colloid degeneration.

Elizabeth P., æt. 81, was admitted under Mr. Davies-Colley for a strangulated femoral hernia, from which she died. There was no history of vomiting or other symptom of disease of the stomach until the onset of the intestinal obstruction four days before her death. At the autopsy no secondary deposits were found. *See Insp.* 1890, No. 390.

**3726a Carcinoma of the Stomach causing Perforation.**

A stomach, laid open to shew the greater part of its wall occupied by a prominent ulcerated growth. Upon the posterior wall and along the greater curvature all the coats

of the organ are invaded by the growth which projects from the serous surface. At about the middle of the greater curvature there is a ragged perforation marked by a blue rod. Histologically the growth is a cylindrical-celled carcinoma.

Samuel A., æt. 42, was admitted under Dr. Taylor for a tumour in the epigastric region and progressive emaciation. He had suffered from occasional attacks of vomiting and hæmatemesis for two years. He died four months after admission. At the autopsy the stomach was found firmly adherent to neighbouring organs, and there was general suppurative peritonitis. There were secondary deposits in the liver and in the mediastinal glands. *See Insp.* 1891, No. 415.

**3731 Sarcoma of the Stomach.**

A stomach from which a portion of the anterior wall has been removed to shew a rounded mass of growth about the size of a pigeon's egg projecting from its posterior wall an inch and a half from the pylorus. Around this mass the walls of the stomach are thickened and infiltrated by growth. The area thus affected extends from the pyloric ring along the lesser curvature to the œsophageal opening, and for six inches along the greater curvature, and is separated by a well-defined margin from the healthy parts beyond. Externally there is a considerable deposit of growth, embedded in which is seen the colon closely adherent to the stomach. Histologically the growth is a sarcoma with round and spindle cells.

Louisa B., æt. 38, was admitted under Dr. Goodhart for vomiting and diarrhœa with distension of the abdomen of four weeks' duration. She died a fortnight after admission. At the autopsy the peritoneal cavity was found to be nearly obliterated by adhesions. There were no secondary deposits. *See Insp.* 1887, No. 116.

# LIST

OF

# GENTLEMEN EDUCATED AT GUY'S HOSPITAL

WHO HAVE PASSED THE

## EXAMINATIONS OF THE SEVERAL UNIVERSITIES, COLLEGES,

&c. &c.,

IN THE YEAR 1892.

---

### University of Oxford.

*Final Examination for the Degrees of Bachelor of Medicine and Surgery.*

E. H. Cartwright.

*First Examination for the Degrees of Bachelor of Medicine and Surgery.*

W. E. Robinson.

---

### University of Cambridge.

*Final Examination for the Medical and Surgical Degree.*

Part I.

W. E. Alston.
J. S. Barnes.
G. T. Birdwood.
R. H. Luce.
H. B. Bolus.
W. S. Frith.
W. Poole.
J. H. Godson.
W. J. Harris.
H. C. Sharp.
G. F. Still.

Part II.

J. S. Barnes.
G. T. Birdwood.
A. M. Daniel.
W. S. Frith.
H. C. Sharp.
W. R. Grove.
C. N. Thomas.
J. D. Thomas.
F. C. Young.
E. Ransome.

*Second Examination for the Medical and Surgical Degrees.*

Part I.

R. J. Rowland.

Part II.

A. P. Beddard.
E. E. Henderson.
T. L. Jackson.
B. W. M. A. Key.
P. R. Lowe.

---

## University of London.

*Examination for the Degree of Doctor of Medicine.*

J. St. T. Clarke.
T. G. Stevens.
A. S. Wohlmann.

---

*Examination for the Degree of Master in Surgery.*

T. B. P. Davies.

*Obtained the Gold Medal.*

F. W. Hall.
†W. G. Rogers.

†*Obtained Marks qualifying for the Medal.*

---

*Examination for the Degree of Bachelor of Surgery.*

First Division.

E. T. E. Hamilton.
C. S. Pantin.
T. R. Taylor.

*Obtained First-Class Honours.*

T. D. Lister.
J. W. F. Jewell.

*Obtained Honours.*

W. Bligh.

Second Division.

F. J. Coleman.
A. W. Sheen.

*Obtained Honours.*

A. M. Daldy.
C. C. Elliott.
S. G. Floyd.
A. J. Sharp.

---

*Final Examination for the Degree of Bachelor of Medicine.*

May.

First Division.

T. Holmes.
P. Lord.
A. W. Sheen.

Second Division.

C. J. Girling.
J. W. F. Jewell.

October.

First Division.

A. M. Daldy.

*Obtained the Gold Medal in Medicine and First-Class Honours in Obstetric Medicine.*

C. C. Elliott.

*Obtained First-Class Honours in Medicine.*

E. T. E. Hamilton.

*Obtained First-Class Honours in Medicine and Honours in Forensic Medicine.*

W. Bligh.

*Obtained Honours in Medicine.*

F. J. Coleman.

*Obtained Honours in Medicine.*

A. J. Sharp.

*Obtained First-Class Honours in Forensic Medicine and Honours in Medicine.*

C. S. Pantin.

*Obtained Honours in Medicine and in Obstetric Medicine.*

S. G. Floyd. | T. R. Taylor.

Second Division.

T. D. Lister.

*Intermediate Examination in Medicine.*

January.

Second Division.

C. E. Carpmael. | G. S. Hovenden.
A. C. Hovenden. | A. Miller.
L. A. Parry.

*Excluding Physiology.*

First Division.

C. R. Colley.

Second Division.

A. Chaning-Pearce. | E. F. H. Hardenberg.
N. Instone.

*Physiology only.*

First Division.

W. E. de Korté.

July.

Honours Examination.

F. J. Steward.

*Obtained the Exhibition and Gold Medal in Chemistry, the Gold Medal in Anatomy, and the Gold Medal in Physiology.*

H. Nolan.

*Obtained the Exhibition and Gold Medal in Materia Medica, First-Class Honours in Chemistry, and Honours in Physiology.*

W. S. Handley.

*Obtained Honours in Physiology and in Organic Chemistry.*

A. Salter.

*Obtained Honours in Physiology.*

Pass List.

First Division.

A. Armer. | E. C. Taylor.

Second Division.

J. J. Coleman.
H. W. Collier.
J. H. Horton.
B. A. Richmond.
P. N. Vellacott.
C. F. Wakefield.

*Excluding Physiology.*

Second Division.

W. M. Price.

*Physiology only.*

First Division.

C. R. Colley. | E. F. H. Hardenberg.

Second Division.

A. Chaning-Pearce. | H. W. Hart.
N. Instone.

---

*Preliminary Scientific (M.B.) Examination.*

January.

Entire Examination.

First Division.

V. E. Collins.

Second Division.

A. J. Wernet.

*Chemistry and Physics only.*

R. T. Fitz-Hugh. | W. E. Waymark.

July.

Honours Examination.

H. J. Starling.

*Obtained Honours in Botany.*

Pass List.

Entire Examination.

Second Division.

H. D. Peile. | W. H. M. Smith.

*Chemistry and Physics only.*

H. Leader.

*Biology only.*

J. D. Bridger.
A. J. Cleveland.
R. T. Fitz-Hugh.
J. A. Glover.
C. R. Hodgson.
N. Lavers.
R. W. Mayston.
G. B. Webb.

---

*Final Examination for the Degree of B.Sc.*

First Division.

G. L. Eastes.

*Obtained Honours in Physiology.*

Second Division.

A. H. Leete.

*Obtained Honours in Physiology.*

B. A. Richmond.

*Obtained Honours in Botany.*

---

*Intermediate (LL.B.) Examination.*

H. Nolan

*Obtained Honours in Jurisprudence.*

---

## University of Durham.

*Examination for the Degree of Doctor of Medicine.*

J. W. Sandoe.

*Second Examination for the Degree of Bachelor of Medicine.*

E. R. Fothergill.
W. F. Miller.
W. H. Peake.
F. W. Rowland.
W. J. Rowland.
F. Zumbado.

*First Examination for the Degree of Bachelor of Medicine.*

*Chemistry and Chemical Physics and Botany.*

W. C. Pakes.
T. B. Poole.

*Chemistry and Chemical Physics.*

O. Beven.

---

## Royal College of Physicians.

*Fellowship.*

Lauriston E. Shaw.

*Final Examination for the Licence.*

January.

H. G. Biddle.
H. B. Bolus.
C. B. Braithwaite.
J. J. Browne.
R. C. Fraser.
W. A. Haslam.
W. A. Higgs.
A. R. Lacey.
P. Lord.
F. D. Lumley.
A. E. Norris.
C. H. D. Ralph.
H. B. Wilkinson.
H. E. Worthington.

April.

G. T. Birdwood.
W. Bligh.
W. F. Colclough.
C. C. Elliott.
J. R. Evans.
E. E. Frazer.
E. H. Greaves.
E. E. B. Landon.
W. D. Loveday.
E. G. March.
P. M. May.
C. G. F. Morice.
J. R. R. Pollock.
H. K. Rayson.
F. G. Saffery.
T. G. Scott.
A. W. Sheen.
G. T. S. Sichel.
F. C. Stearn.
R. L. Wason.

July.

H. Elliot-Blake.
H. St. J. Fraser.
E. B. Hartnell.
J. H. Harvey.
Guy Mackeson.
F. A. Osborn.
E. W. Wheatcroft.

October.

C. M. Anderson.
R. H. W. H. Brabant.
H. A. Bryant.
E. H. Cartwright.
F. P. S. Cresswell.
C. M. Fleury.
S. G. Floyd.
R. Gillbard.
C. G. Gregar.
E. Huntley.
A. F. Jago.
T. D. Lister.
S. J. Roberts.
F. A. Robinson.
E. M. Rooke.

---

## Royal College of Surgeons.

*Final Examination for the Fellowship.*

J. Fawcett.
E. T. E. Hamilton.
H. M. Jordan.
A. T. Rake.
A. W. Sheen.

*First Examination for the Fellowship.*

C. H. Bryant.
G. L. Eastes.
G. E. Manning.
W. G. Mumford.
L. A. Parry.

*Final Examination for the Membership.*

January.

H. G. Biddle.
H. B. Bolus.
C. B. Braithwaite.
J. J. Browne.
R. C. Fraser.
W. A. Haslam.
W. A. Higgs.
A. R. Lacey.
P. Lord.
F. D. Lumley.
A. E. Norris.
C. H. D. Ralph.
H. B. Wilkinson.
H. E. Worthington.

April.

G. T. Birdwood.
W. Bligh.
W. F. Colclough.
C. C. Elliott.
J. R. Evans.
E. E. Frazer.
E. H. Greaves.
E. E. B. Landon.
W. D. Loveday.
E. G. March.
P. M. May.
C. G. F. Morice.
J. R. R. Pollock.
H. K. Rayson.
F. G. Saffery.
T. G. Scott.
A. W. Sheen.
G. T. S. Sichel.
F. C. Stearn.
R. L. Wason.

July.

H. Elliot-Blake.
H. St. J. Fraser.
E. B. Hartnell.
J. H. Harvey.
Guy Mackeson.
F. A. Osborn.
E. W. Wheatcroft.

October.

C. M. Anderson.
R. H. W. H. Brabant.
H. A. Bryant.
E. H. Cartwright.
F. P. S. Cresswell.
C. M. Fleury.
S. G. Floyd.
R. Gillbard.
C. G. Gregar.
E. Huntley.
A. F. Jago.
T. D. Lister.
S. J. Roberts.
F. A. Robinson.
E. M. Rooke.

*Final Examination for the Licence in Dental Surgery.*

F. P. Allworth.
S. R. Apthorpe.
F. J. Blewitt.
P. Harrison.
A. R. Henry.
J. S. Sewill.
H. B. Stoner.
H. A. Turner.
R. E. Woodcock.

## Physical Society.

*Session* 1892-3.

**Honorary President.**—S. WILKS, M.D., F.R.S.

**Presidents.**

Messrs. W. Bligh, M.B., B.S., A. M. Daldy, M.B., B.S., L. E. Every-Clayton, E. T. E. Hamilton, M.B., B.S., B.Sc., F. G. Hopkins, B.Sc., J. A. Howard, B.Sc., J. B Leathes, B.A., T. D. Lister, M.B., B.S., C. S. Pantin, M.B., B.S., C. E. Salter, M.B., B.S., A. J. Sharp, M.B., B.S., A. W. Sheen, M.B., B.S., W. K. Steele, H. M. Stewart, B.A., T. R. Taylor, M.B., B.S., B.Sc., C. F. Wakefield.

**Exhibits Committee.**

Messrs. A. M. Daldy, M.B., B.S., L. E. Every-Clayton, J. Eyre, H. P. Ferraby, E. T. E. Hamilton, M.B., B.S., B.Sc., J. A. Howard, B.Sc., C. F. Wakefield.

**Photographic Committee.**

Messrs. P. Hacquoil, A. C. Hovenden, G. S. Hovenden, T. S. Jackson, W. E. de Korté, E. A. Peters, B.A., H. A. Hinds.

**Honorary Secretaries.**—L. A. DUNN, M.S., H. J. CAMPBELL, M.D.

PRIZEMEN FOR THE SESSION 1892-3.

The Society's Prizes, First, £7, was awarded to Mr. J. A. Howard, B.Sc., for his Paper on "Uræmia."

The Speaking Prize of £4 for Senior Students was given to Mr. D. M. Beddoe.

An Extra Prize of £5, given by the Treasurer, was awarded to Mr. L. E. Every-Clayton, for his Paper on "The Pathology of Fœtal Relics."

---

## CLINICAL APPOINTMENTS HELD IN THE YEAR 1892.

HOUSE PHYSICIANS.

| | | |
|---|---|---|
| D. S. Long. | J. Young. | J. S. Richards. |
| W. Winslow. | C. E. Salter. | F. M. Turner. |
| A. Thomas. | J. W. F. Jewell. | F. C. Young. |
| D. W. Samways. | | |

HOUSE SURGEONS.

| | | |
|---|---|---|
| R. S. Freeland. | F. S. Wood. | H. Hodgson. |
| W. G. Rogers. | H. E. Durham. | T. B. P. Davies. |
| A. T. Rake. | W. Carling. | A. H. Godson. |
| H. B. Rygate. | | |

OBSTETRIC RESIDENTS.

| | | |
|---|---|---|
| J. Young. | A. S. Wohlmann. | A. Thomas. |
| A. M. Daldy. | C. E. Salter. | C. E. Pollock. |
| G. Sichel. | E. E. B. Landon. | H. B. Bolus. |
| W. H. Fisher. | R. L. Wason. | T. B. P. Davies. |
| H. W. Webber. | | |

SURGEONS' DRESSERS.

E. Hunt.
A. V. Chapman.
E. Huntley.
F. C. Stearn.
S. G. Floyd.
F. Hazell.
H. P. Bolus.
W. Bligh.
D. A. W. Martin.
G. A. Skinner.
W. J. Harris.
R. E. G. Phillips.
A. Shillitoe.
H. A. Hinds.
J. W. H. Eyre.
E. Reid.
R. C. Kirkby.
W. E. de Korté.
J. H. Compton.
A. L. Roper.
A. J. Sharp.
G. Lawrence.
E. E. S. Silver.
A. G. Cooley.
W. R. Grove.
G. H. Steele.
A. B. Creak.
H. P. Barker.
A. P. Allan.

H. D. Eccles.
F. J. Coleman.
C. E. Pollock.
W. J. Johnson.
T. S. Jackson.
P. E. Bearblock.
A. H. Card.
F. A. Robinson.
J. W. Eastment.
H. Hearnden.
H. Meyrick-Jones.
G. E. Lockyer.
H. Cooper.
W. Poole.
H. C. Sharp.
F. H. Edwards.
J. J. E. Biggs.
F. G. M. Philps.
W. H. Jewell.
P. H. Hacquoil.
F. G. Hopkins.
H. A. Munro.
S. Infield.
J. G. B. Coleman.
G. Jimenez.
N. Instone.
C. F. Wakefield.
V. Pendred.
E. E. W. Roe.

J. H. Godson.
E. J. H. Budge.
E. S. Dukes.
R. H. Luce.
F. W. Wilson.
W. E. Alston.
J. R. R. Pollock.
F. N. Day.
F. P. Bush.
J. W. Taylor.
G. H. Field.
W. C. C. Park.
E. L. Adams.
S. C. Cresswell.
G. F. Still.
E. S. Tuck.
J. H. Joyce.
D. M. Beddoe.
S. Croneen.
T. D. Manning.
C. S. Pantin.
J. B. Leathes.
A. H. Trevor.
J. J. Culmer.
E. Ransome.
C. M. Greenway.
C. E. Cooper.
C. H. Harding.
P. McK. Wilmot.

CLINICAL ASSISTANTS.

A. M. Daldy.
H. E. Durham.
E. Huntley.
W. Bligh.
W. S. Frith.
W. J. Johnson.

A. T. Rake.
F. M. Turner.
C. C. Elliott.
J. D. Thomas.
G. T. Birdwood.
R. H. Luce.

T. R. Taylor.
E. T. E. Hamilton.
R. L. Wason.
F. J. Coleman.
H. Cooper.
J. A. Howard.

DRESSERS IN THE EYE WARDS.

C. Braithwaite.
A. W. Sheen.
P. M. May.
E. T. E. Hamilton.
A. M. Daldy.
T. D. Lister.
J. R. Evans.

C. G. F. Morice.
J. O. Williams.
A. G. Buchanan.
E. E. B. Landon.
F. P. S. Cresswell.
J. H. Muncaster.
H. C. Sharp.

H. B. Wilkinson.
F. C. Young.
T. R. Taylor.
W. E. Alston.
F. W. Wilson.
J. S. Barnes.

DENTAL SURGEONS' DRESSERS.

A. W. Sheen.
H. B. Bolus.
A. L. Roper.
E. S. Dukes.
H. Cooper.
C. E. Salter.

T. I. Mills.
D. S. Long.
J. H. Godson.
W. Poole.
G. Hovenden.

R. H. Luce.
N. Instone.
E. T. E. Hamilton.
J. H. Muncaster.
C. E. Cooper.

MEDICAL WARD CLERKS.

D. F. Roberts.
P. McK. Wilmot.
F. G. M. Philps.
J. H. Compton.
J. G. B. Coleman.
J. H. Joyce.
A. L. Roper.
R. H. Moon.
H. M. Stewart.
A. B. Creak.
W. R. Grove.
A. J, Collis.
V. Pendred.
C. R. Lucas.
H. G. T. Major.
A. H. Leete.
G. E. Manning.
J. C. V. Wilkins.
L. C. Burrell.
F. Cowan.
A. P. Rainbird.
E. F. H. Hardenberg.
H. Hewetson.
C. R. Colley.
E. A. Peters.
S. S. Wallis.
J. W. Sweetlove.
C. L. Hopkins.
J. J. Culmer.
P. H. Hacquoil.
J. W. H. Eyre.
C. H. Harding.
E. Reid.
C. E. Cooper.
E. Ransome.
G. Jimenez.
H. W. Graham.
J. H. Bettington.
L. Slater.
H. B. Barker.
H. W. Beach.
E. E. W. Rowe.
W. J. Burroughs.
A. C. Hovenden.
R. H. Fagge.
R. D. Evans.
T. Burrow.
C. S. Bond.
L. A. Parry.
L.E.V.Every-Clayton
F. Zumbado.
H. T. Hinton.
W. C. Pitt.
W. D. Macdonald.
C. E. Carpmael.
T. P. King.
S. Infield.
F. H. Edwards.
J. J. E. Biggs.
D. M. Beddoe.
W. E. de Korté.
H. A. Munro.
H. Davies.
J. W. Culmer.
A. P. Allan,
A. G. Cooley.
J. B. Winter.
A. Chaning-Pearce.
J. A. Howard.
C. W. Moorshead.
E. H. Drake.
G. S. Hovenden.
A. J. Mathison.
W. W. Henson.
T. M. Nicholson.
J. R. Steinhaeuser.
W. C. Lawton.
A. K. Matthews.
W. Peake.
A. Miller.
F. A. Evison.
S. G. Graham.
C. A. Ensor.
E. C. Taylor.

ASSISTANT SURGEONS' DRESSERS.

D. A. W. Martin.
A. H. Leete.
A. J. Sharp.
F. Cowan.
W. C. C. Park.
G. E. Lockyer.
G. E. Manning.
A. H. Trevor.
E. E. S. Silver.
G. Lawrence.
R. C. Kirbky.
E. H. Drake.
E. A. Peters.
T. Burrow.
W. C. Lawton.
P. McK. Wilmot.
J. W. Sweetlove.
G. H. Steele.
W. C. Pitt.
C. M. Greenway.
H. W. Graham.
A. E. Phillips.
H. Davies.
C. R. Lucas.
T. S. Biggs.
R. H. Moon.
J. H. Bettington.
J. C. Constable.
G. Jimenez.
J. C. V. Wilkins.
W. J. Harris.
H. Meyrick-Jones.
A. J. Collis.
A. P. Allan.
J. W. Eastment.
E. S. Tuck.
J. B. Leathes.
A. C. Hovenden.
A. J. Mathison.
A. Alexander.
R. D. Evans.
S. Infield.
R. Serjeant.
C. E. Carpmael.
C. L. Hopkins.
S. S. Wallis.
C. R. Colley.
T. P. King.
J. H. Busteed.
H. Pern.
J. W. Culmer.
T. H. Green.
B. G. Reynolds.
L. Bensted.
W. F. Miller.
E. C. Roberts.
J. B. Winter.
C. S. Pantin.
J. A. Howard.
H. Hearnden.
V. Pendred.
W. Poole.
F. G. Hopkins.
T. D. Manning.
W. H. Jewell.
G. S. Hovenden.
R. Ingram.
H. T. Hinton.
R. H. Fagge.
N. Instone.
S. Hughes.
H. A. Duffet.

ASSISTANT SURGEONS' DRESSERS (*continued*).

W. D. Macdonald.
F. A. Evison.
E. S. Hoare.
A. Miller.
F. Brereton.
F. H. Cloud.
A. Chaning-Pearce.
S. Copley.
H. W. Beach.
H. E. Cock.
H. M. Stewart.
A. C. Fry.

EXTERN OBSTETRIC ATTENDANTS.

A. Shillitoe.
W. J. Johnson.
J. W. Culmer.
L. Slater.
E. A. Peters.
W. C. Lawton.
W. Poole.
W. J. Harris.
F. N. Day.
P. E. Bearblock.
N. Instone.
W. F. Miller.
J. R. Evans.
J. B. Leathes.
J. H. Godson.
J. H. Busteed.
J. J. Culmer.
A. H. Trevor.
A. P. Allan.
P. McK. Wilmot.
T. H. B. Yorath.
P. Hacquoil.
R. H. Hayes.
A. H. Card.
F. J. Coleman.
R. H. Fagge.
W. H. Jewell.
H. M. Stewart.
L. C. Burrell.
H. Cooper.
G. F. Still.
E. Hunt.
H. D. Eccles.
C. E. Covernton.
E. S. Roberts.
J. W. H. Eyre.
G. Lawrence.
G. H. Field.
H. E. Cock.
G. E. Lockyer.
C. H. Harding.
E. E. W. Roe.
E. S. Hoare.
E. S. Tuck.
F. G. M. Philps.
C. C. Jenkins.
F. A. Robinson.
F. Hazell.
F. P. Bush.
A. G. Cooley.
H. W. Beach.
W. H. Fisher.
W. C. C. Park.
G. H. Steele.
A. V. Chapman.
W. J. Burroughs.
H. A. Duffet.
T. D. Manning.
F. G. Hopkins.
J. W. Taylor.
E. C. Taylor.
A. Alexander.
H. Hearnden.
S. Infield.
A. J. Collis.
R. C. Kirkby.
J. J. E. Biggs.
R. Serjeant.
J. C. Peatson.

SURGICAL WARD CLERKS.

W. D. Macdonald.
L. Bensted.
W. L. Garner.
W. C. Pitt.
C. S. Bond.
T. P. King.
T. M. Nicholson.
C. E. Carpmael.
A. Miller.
H. Hewetson.
C. H. Fagge.
E. C. Taylor.
H. Pern.
W. E. Pain.
S. G. Graham.
A. K. Matthews.
L. E. V. Every-Clayton.
T. H. Green.
T. M. Thomas.
J. G. O. Lane.
W. A. L. Smith.
C. H. Bryant.
E. C. B. Ibotson.
J. H. Horton.
W. S. Handley.
W. A. Carden.
F. Loud.
W. L. B. Davies.
S. S. Wallis.
A. C. Fry.
R. H. Moon.
S. Hughes.
W. W. Henson.
R. D. Evans.
E. S. Roberts.
C. R. Colley.
E. F. H. Hardenberg.
J. C. Constable.
J. H. Busteed.
H. F. Turner.
D. W. Thomas.
T. S. Biggs.
L. A. Parry.
W. F. Miller.
F. Zumbado.
C. A. Ensor.
H. M. Wise.
F. S. Brereton.
A. V. Clarke.
W. G. Mumford.
H. Nolan.
F. J. Steward.
M. P. Jones.
C. J. Kearney.
H. D. Packer.
W. T. Byford.
F. A. Evison.
F. H. L. Cloud.
T. Burrow.
H. E. Cock.
H. Davies.
J. W. Sweetlove
A. P. Rainbird.
J. C. Peatson.
R. Serjeant.

SURGICAL WARD CLERKS' (*continued*).

H. E. Cock.
P. J. Curtis.
S. Copley.
J. R. Steinhaeuser.
H. J. F. Bourne.
W. H. Peake.
C. E. Covernton.
B. G. Reynolds.
A. E. Philipps.
E. Coleman.
H. A. Moffat.
H. W. Collier.
J. J. Coleman.
A. Armer.
A. Salter.
C. M. Beadnell.
E. E. Henderson.
W. E. Robinson.
B. W. M. A. Key.

ASSISTANT SURGEONS' CLERKS.

G. Lawrence.
A. J. Mathison.
C. L. Hopkins.
E. S. Hoare.
C. S. Bond.
F. A. Evison.
E. F. H. Hardenberg.
B. P. O'Neill.
R. H. Hayes.
M. E. Tresidder.
L. H. Y. Stephen.
E. E. Henderson.
C. H. Harding.
R. H. Fagge.
C. E. Cooper.
T. M. Nicholson.
B. W. M. A. Key.
H. M. Stewart.
J. C. Constable.
S. Rivers.
D. W. Thomas.
J. G. O. Lane.
A. P. Rainbird.
H. W. Beach.
F. H. L. Cloud.
E. S. Roberts.
W. L. Garner.
J. C. Peatson.
G. Jimenez.
F. Loud.
P. E. Tresidder.
J. Hora.

AURAL SURGEONS' DRESSERS.

C. E. Salter.
W. Carling.
J. O. Williams.
G. Jimenez.
E. H. Drake.
J. R. R. Pollock.
E. S. Roberts.
H. B. Bolus.
E. E. B. Landon.
H. B. Wilkinson.
H. J. L. Wales.
A. Allport.

ASSISTANT PHYSICIANS' CLERKS.

C. R. Lucas.
H. Cooper.
T. D. Manning.
T. P. King.
W. C. Lawton.
J. G. B. Coleman.
V. Pendred.
T. S. Robson.
C. W. Moorshead.
C. S. Bond.
A. J. Mathison.
T. Burrow.
G. T. Birdwood.
C. L. Hopkins.
R. C. Kirkby.
W. W. Henson.
L. C. Burrell.
E. C. B. Ibotson.
A. J. Collis.
J. A. Howard.
J. C. V. Wilkins.
C. C. Jenkins.
W. J. Burroughs.
E. H. Drake.
J. B. Leathes.
J. D. Thomas.
A. H. Leete.
J. S. Barnes.
S. Worts.
E. S. Dukes.
A. Allport.
A. Chaning-Pearce.
W. W. Henson.
F. Cowan.
R. D. Evans.
R. H. Fagge.

POST-MORTEM CLERKS.

J. H. Bettington.
A. Chaning-Pearce.
W. C. Pitt.
S. G. Floyd.
M. Craig.
W. J. Johnson.
S. C. Cresswell.
J. W. Ensor.
H. M. Stewart.
F. P. Bush.
F. P. S. Cresswell.
A. V. Chapman.
E. Ransome.
J. W. Taylor.
W. C. C. Park.
A. Shillitoe.
W. R. Grove.
E. Huntley.
W. A. Haslam.
F. Hazell.
J. H. Godson.
T. D. Manning.

OBSTETRIC WARD CLERKS.

| | | |
|---|---|---|
| C. G. Gregar. | T. I. Mills. | G. Sichel. |
| J. R. Evans. | C. M. Greenway. | J. C. V. Wilkins. |
| F. N. Day. | A. H. Card. | S. G. Floyd. |
| A. Shillitoe. | E. S. Dukes. | W. Poole. |

OBSTETRIC OUT-PATIENTS' CLERKS.

| | | |
|---|---|---|
| E. A. Peters. | W. R. Grove. | C. C. Elliott. |
| F. P. S. Cresswell. | T. D. Lister. | L. Slater. |
| F. Cowan. | J. D. Thomas. | W. D. Macdonald. |
| H. G. T. Major. | A. H. Card. | C. L. Hopkins. |
| J. O. Williams. | C. C. Jenkins. | E. L. Adams. |
| W. C. C. Park. | W. Poole. | H. C. Sharp. |
| H. P. Barker. | A. R. Hitchfield. | T. H. B. Yorath. |
| E. Huntley. | J. B. Winter. | F. Hazell. |

DRESSERS IN THE THROAT DEPARTMENT.

| | | |
|---|---|---|
| A. Reeves. | W. Winslow. | G. Mackeson. |
| G. F. Still. | J. J. Culmer. | A. Allport. |
| E. G. March. | J. W. H. Eyre. | H. J. L. Wales. |
| F. M. Turner. | E. S. Dukes. | A. P. Allan. |
| C. C. Elliott. | W. Bligh. | F. P. S. Cresswell. |
| E. T. E. Hamilton. | | |

CLERKS IN THE ELECTRICAL DEPARTMENT.

| | | |
|---|---|---|
| G. F. Still. | F. H. Edwards. | H. A. Munro. |
| G. A. Skinner. | C. E. Carpmael. | P. McK. Wilmot. |
| F. G. Saffrey. | J. G. B. Coleman. | L. Bensted. |
| A. H. Card. | W. D. Macdonald. | E. C. B. Ibotson. |
| G. L. Eastes. | C. G. F. Morice. | J. S. Barnes. |
| G. Field. | S. G. Floyd. | E. A. Peters. |

CLERKS TO THE ANÆSTHETISTS.

| | | |
|---|---|---|
| E. Huntley. | J. R. R. Pollock. | R. L. Wason. |
| W. Bligh. | J. D. Thomas. | C. C. Elliott. |
| P. Lord. | F. P. S. Cresswell. | W. A. Haslam. |
| G. T. Birdwood. | C. N. Thomas. | H. B. Wilkinson. |
| E. H. Cartwright. | P. McK. Wilmot. | T. D. Lister. |
| J. W. H. Eyre. | F. J. Coleman. | C. R. Colley. |
| C. G. F. Morice. | H. C. Sharp. | |

## DENTAL SCHOOL.

### APPOINTMENTS HELD DURING THE YEAR 1892.

DENTAL HOUSE SURGEONS.

F. W. Ta'Bois. | H. B. Stoner.

ASSISTANT DENTAL HOUSE SURGEONS.

| | | |
|---|---|---|
| F. J. Blewett. | S. Keele. | R. E. Woodcock. |
| S R. Apthorpe. | | |

DEMONSTRATORS IN THE CONSERVATION ROOM.

A. R. Henry.
H. Turner.
G. S. F. Manton.
R. E. Woodcock.
F. E. Pritchard.
F. H. Wallis.
F. P. Allworth.
W. H. Saul.

CURATORS OF THE LABORATORY.

G. S. F. Manton.
W. R. Butler.
F. E. Pritchard.
J. B. Parfitt.

ASSISTANT DEMONSTRATOR OF DENTAL MICROSCOPY.

S. R. Apthorpe.

DRESSERS IN THE EXTRACTION ROOM.

E. Morgan.
W. R. Wood.
C. Keay.
F. H. Carpenter.
S. H. Longhurst.
L. F. Morris.
H. Cook.
J. Plomley.
T. S. Davison.
W. H. Pilcher.
J. W. Pare.
F. C. Constant.
R. Thornton.
E. Picnot.
E. W. Stabb-Johnson.
F. E. L. Lambert.
W. J. O. Dawson.
J. B. Parfitt.
E. V. Knight.
C. H. Charter.
C. H. Oram.
P. F. Henry.
M. N. Mitchener.
J. Pilcher.
G. B. Webb.
A. H. H. Huckle.
T. Godfrey.
G. H. Bowden.
E. Hutson.
W. H. Trick.
F. C. Bromley.
W. H. Lyne.
S. M. Carpenter.
G. Whitworth.
E. Clayton.
W. H. Lyne.
W. E. Hills.
A. Cahill.
G. W. Jones.
S. Harrison.
A. J. Tyrrell.
F. Mühlenkamp.
G. N. Willis.
U. E. Cave.

DRESSERS IN THE GAS ROOM.

F. H. Wallis.
J. M. Tarachand.
A. V. Brimmer.
G. S. F. Manton.
G. H. Williams.
F. C. Bromley.
F. E. L. Lambert.
C. Read.
E. V. Knight.
S. Keele.
S. R. Apthorpe.
W. H. Lyne.
F. E. Pritchard.
F. P. Allworth.
G. Whitworth.
P. F. Henry.
F. H. Carpenter.
R. E. Woodcock.
W. H. Saul.
C. H. Oram.
L. G. Musgrave.
C. Keay.
J. B. Parfitt.
G. B. Webb.
W. J. O. Dawson.

# GUY'S HOSPITAL.

## MEDICAL AND SURGICAL STAFF.

1893.

**Consulting Physicians.**

S. Wilks, M.D., LL.D., F.R.S.
F. W. Pavy, M.D., LL.D., F.R.S.

**Consulting Obstetric Physicians.**

H. Oldham, M.D.
J. Braxton Hicks, M.D., F.R.S.

**Physicians.**

P. H. Pye-Smith, M.D., F.R.S.
Frederick Taylor, M.D.
J. F. Goodhart, M.D.
W. Hale White, M.D.

**Assistant Physicians.**

G. Newton Pitt, M.D.
E. C. Perry, M.D.
L. E. Shaw, M.D.
J. W. Washbourn, M.D.

**Obstetric Physician.**

A. L. Galabin, M.D.

**Assistant Obstetric Physician.**

P. Horrocks, M.D.

**Dental Surgeon.**

F. Newland-Pedley, Esq.

**Senior Assistant Dental Surgeon.**

W. A. Maggs, Esq.

**Medical Registrar.**

J. H. Bryant, M.D.

**Consulting Surgeons.**

J. Birkett, Esq.
Thomas Bryant, M.Ch.

**Consulting Ophthalmic Surgeon.**

Charles Bader, Esq.

**Surgeons.**

Arthur E. Durham, Esq.
H. G. Howse, M.S.
N. Davies-Colley, M.C.
R. Clement Lucas, B.S.

**Assistant Surgeons.**

C. H. Golding-Bird, M.B.
W. H. A. Jacobson, M.Ch.
Charters J. Symonds, M.S.
W. Arbuthnot Lane, M.S.

**Ophthalmic Surgeon.**

C. Higgens, Esq.

**Assistant Ophthalmic Surgeon.**

W. A. Brailey, Esq.

**Aural Surgeon.**

W. Laidlaw Purves, Esq.

**Instructors in Anæsthetics.**

Tom Bird, Esq.
F. Silk, M.D.

**Surgical Registrar.**

J. McD. Gill, M.D.

---

**Curator of the Museum.**

Dr. Shaw.

**Lying-in Charity.**

Dr. Galabin and Dr. Horrocks.

**Dean of the Medical School.**

Dr. Shaw.

**Warden of the College.**

Mr. L. A. Dunn, M.S.

## LECTURERS AND DEMONSTRATORS.

| | |
|---|---|
| *Clinical Medicine* ... ... ... ... | The Physicians and Assistant Physicians. |
| *Clinical Surgery* ... ... ... ... | The Surgeons and Assistant Surgeons. |
| *Medicine*... ... ... ... ... ... | Dr. Pye-Smith and Dr. Taylor. |
| *Practical Medicine* ... ... ... | Dr. Bryant. |
| *Surgery* ... ... ... ... ... ... | Mr. Howse and Mr. Davies-Colley. |
| *Practical Surgery*... ... ... ... | Mr. Symonds and Mr. Lane. |
| *Operative Surgery*... ... ... ... | Mr. Symonds. |
| *Midwifery and Diseases of Women* | Dr. Galabin. |
| *Practical Obstetrics* ... ... ... | Dr. Horrocks. |
| *Mental Diseases* ... ... ... ... | Dr. Savage. |
| *Ophthalmic Surgery* ... ... ... | Mr. Higgens and Mr. Brailey. |
| *Dental Surgery* ... ... ... ... | Mr. Newland-Pedley. |
| *Aural Surgery* ... ... ... ... | Mr. Laidlaw Purves. |
| *Diseases of the Skin* ... ... ... | Dr. Perry. |
| *Diseases of the Throat* ... ... ... | Mr. Symonds. |
| *Electro-Therapeutics* ... ... ... | Dr. Washbourn. |
| *Anæsthetics* ... ... ... ... ... | Mr. Tom Bird and Dr. Silk. |
| *Hygiene and Public Health*... ... | Mr. Turner. |
| *Pathology* ... ... ... ... ... | Dr. Pitt. |
| *Morbid Anatomy* ... ... ... ... | Dr. Pitt, Dr. Perry, and Dr. Shaw. |
| *Morbid Histology* ... ... ... ... | Dr. Pitt and Mr. Lane. |
| *Bacteriology* ... ... ... ... ... | Dr. Washbourn. |
| *Medical Jurisprudence*... ... ... | Dr. Stevenson. |
| *Anatomy*... ... ... ... ... ... | Mr. Lucas and Mr. Jacobson. |
| *Practical Anatomy* ... ... ... | Mr. Dunn, Mr. Targett, and Mr. Fripp. |
| *Physiology* ... ... ... ... ... | Mr. Golding-Bird, Dr. Washbourn, and Dr. Starling. |
| *Histology and Practical Physiology* | Mr. Tubby, Dr. Campbell, and Dr. Fawcett. |
| *Materia Medica, Pharmacology, and Therapeutics* ... ... ... | Dr. Hale White. |
| *Practical Pharmacy* ... ... ... | The Hospital Dispenser. |
| *Chemistry* ... ... ... ... ... | Dr. Stevenson. |
| *Practical Chemistry* ... ... ... | Mr. Groves. |
| *Experimental Physics* ... ... ... | Professor Reinold. |
| *Biology* ... ... ... ... ... ... | Mr. Beddard and Dr. Campbell. |

---

The Hospital contains 695 beds, of which 500 are in constant occupation Special Classes are held for Students preparing for the Examinations of the University of London, and other Higher Examinations.

## APPOINTMENTS.

All Hospital Appointments are made strictly in accordance with the merits of the Candidates, and without extra payment.

## ENTRANCE SCHOLARSHIPS IN SEPTEMBER, 1892.

Two Open Scholarships in Arts, one of the value of 100 guineas open to candidates under 20 years of age; and one of 50 guineas open to candidate under 25 years of age. Two Open Scholarships in Science, one of the value of 125 guineas, and another of 50 guineas, open to candidates under 25 years of age.

## PRIZES

Are awarded to Students in their various years, amounting in the aggregate to more than £450.

## DENTAL SCHOOL.

The Dental School is attached to the Hospital, which affords to Students all the instruction required for a Licence in Dental Surgery.

## COLLEGE.

The Residential College accommodates about 50 Students in addition to the Resident Staff of the Hospital, and contains a large Dining Hall, Reading Rooms, and Gymnasium for the use of the STUDENTS' CLUB.

---

For Prospectus and further information apply to the Dean, Dr. SHAW, Guy's Hospital, London, S.E.

# GUY'S HOSPITAL.

## DENTAL SCHOOL.
1893.

### Attendance of Assistant Dental Surgeons.

*Mornings.*

Mr. Maggs, L.R.C.P., M.R.C.S., L.D.S.Eng.
Mr. Mansbridge, L.R.C.P., M.R.C.S., L.D.S.Eng.
Mr. Harry Pillin, L.D.S.Eng.
Mr. G. O. Richards, M.R.C.S., L.D.S.Eng.
Mr. Rouw, L.R.C.P., M.R.C.S., L.D.S.Eng.
Mr. H. Stoner, L.D.S.Eng.

*Afternoons.*

Mr. Badcock, L.R.C.P., M.R.C.S., L.D.S.Eng.
Mr. A. E. Baker, L.R.C.P., M.R.C.S., L.D.S.Eng.
Mr. J. O. Butcher, L.D.S.Eng.
Mr. Traer Harris, M.R.C.S., L.S.A., L.D.S.Eng.
Mr. Hopson, L.D.S.Eng.

The *Dental Surgeon*, Mr. Newland-Pedley, F.R.C.S., L.D.S.Eng., attends on *Tuesdays* in the Extraction Room to see special cases.

### Anæsthetists.

Dr. Cock, M.S.
Dr. J. F. Silk.
Dr. Lancaster.
Mr. Fripp, M.B., B.S.
Mr. W. J. Scott, M.B., B.S.
Mr. C. J. Ogle, M.R.C.S., L.S.A.

## LECTURES AND DEMONSTRATIONS.

### Winter Session.

| | |
|---|---|
| **Dental Surgery** . . . . | Mr. Newland-Pedley. |
| **Dental Anatomy and Physiology** . | Mr. Maggs. |
| **Metallurgy** . . . . . | Mr. Groves, F.R.S. |

### Summer Session.

| | |
|---|---|
| **Dental Mechanics** . . . . | Mr. Richards. |
| **Dental Microscopy** . . . | Mr. Mansbridge. |
| **Operative Dental Surgery** . . | Mr. Badcock. |
| **Demonstrations of Dental Mechanics** | Mr. Pillin. |
| **Dental Tutor** . . . . . | Mr. Rouw. |

**The Dental Department** of Guy's Hospital was organised as a complete School of Dental Surgery at the beginning of the Winter Session, 1889. In addition to the accommodation which already existed in the Out-patient Department for the extraction of teeth, the Governors erected special buildings—a Conservation Room, in which the operations of conservative dentistry were carried out, and a Dental Laboratory, in which Students received instruction in the construction and adjustment of artificial dentures for patients under their care. The rapid growth of the School soon necessitated increased accommodation.

**The New Dental School Building** is of considerable extent and supplies all that is requisite for the practice and teaching of Dental Surgery. The new Conservation Room contains fifty Modern Operating Chairs, and it is open all day for practice; and the Special Lectures are arranged to harmonise with the General Lectures.

**The New Dental Laboratory** is 65 ft. long. There are seats for thirty students, who are allowed to construct and fit dentures and correction-plates for patients, thus gaining valuable experience in taking models, making "bites," and adjusting artificial teeth in the mouth. Suitable cases are found in the Hospital, and the work done is absolutely free of charge. Practical instruction is given to supplement the Course of Lectures on Dental Mechanics, and Demonstrations are held on the construction of dental splints, obturators, and vela, used in treating patients in the wards, where there are always interesting cases of surgical disorders, complicated with dental lesions.

There is an **Extraction Room** for ordinary cases, and a **Gas Room** for operations under Anæsthetics, which are administered daily to such patients as the dental officers recommend. Cases suitable for conservative treatment are transferred to the Conservation Room at the discretion of the Assistant Dental Surgeons.

**The Teaching Power** of the School has also been augmented by the appointment of additional Assistant Dental Surgeons, who attend in the afternoons.

**Every Facility** is afforded to students desiring to take medical qualifications in addition to the L.D.S., and in their case the dental curriculum can be spread over the whole general course.

**Continuous Instruction** is afforded by means of a separate morning and afternoon staff of Dental Officers. The lectures are

given at hours convenient to students. Ten clinical demonstrations are given weekly by members of the Dental Staff.

**A Clinical Demonstration** is given at 11 a.m. by the Assistant Dental Surgeon for the day on one of the various methods of filling, the preparation of roots for pivots, and the adaptation of porcelain and metal crowns to broken-down teeth.

An **Entrance Scholarship** and **Prizes** are offered for competition. For First Year's Students, a Prize of £10; for Second Year's Students, Prizes of £15 and £10—the latter being given for Operative Dental Surgery.

---

**Fees.**—The Fee for the two years' Hospital Practice required by the Examining Bodies for the L.D.S., including Lectures, is 30 Guineas. Dental Students are also required to receive two years' instruction in a General Hospital, the Fee for which in Guy's Hospital Medical School is £60, with a reduction of 12 Guineas in the case of those Students who have obtained certificates of instruction in Chemistry, Practical Chemistry and Materia Medica. No arrangements are made for short periods of Instruction or Special Courses of Lectures.

Further particulars with Prospectus may be obtained from the Dean, Dr. L. E. Shaw, Guy's Hospital, S.E.

# ASTLEY COOPER PRIZE.

## The next Triennial Prize of Three Hundred Pounds,

*Under the Will of the late* SIR ASTLEY P. COOPER, Bart.,

WILL BE AWARDED TO

THE AUTHOR OF THE BEST ESSAY OR TREATISE ON

"*The Anatomical Distribution of the Lymphatic Vessels and the Physical Forces concerned in the movement of the Lymph.*"

THE Conditions annexed by the Testator are, "That the Essays or "Treatises to be written for such Prize shall contain original experi- "ments and observations, which shall not have been previously pub- "lished, *and that each Essay or Treatise shall* (as far as the subject "shall admit of) *be illustrated by preparations and by drawings*, which "preparations and drawings shall be added to the Museum of Guy's "Hospital, and shall, together with the Work itself and the sole and "exclusive interest therein and the copyright thereof, become hence- "forth the property of that Institution, and shall be relinquished and "transferred as such by the successful candidate."

And it is expressly declared in the Will "That no Physician or "Surgeon, or other officer for the time being, of Guy's Hospital or of "St. Thomas's Hospital, in the Borough of Southwark, nor any person "related by blood or affinity to any such Physician or Surgeon, for "the time being, or to any other Officer for the time being in either "of the said Hospitals, shall at any time receive or be entitled to "claim the Prize." But, with the exceptions here referred to, this Prize is open for competition to the whole world.*

Candidates are informed that their Essays, either written in the English Language, or, if in a Foreign Language, accompanied by an English translation, must be sent to Guy's Hospital on or before January 1st, 1895, addressed to the Physicians and Surgeons of Guy's Hospital.

Each Essay or Treatise must be distinguished by a Motto, and be accompanied by a sealed envelope containing the name and address of the Writer. None of the envelopes will be opened except that which accompanies the successful Treatise. The unsuccessful Essays or Treatises, with the illustrative preparations or drawings, will remain at the Museum of Guy's Hospital until claimed by the respective writers or their agents.

* The Prize cannot be awarded to any Essay which is the joint production of two or more authors.

# GUY'S HOSPITAL REPORTS FOR 1891.

*The Forty-eighth Volume, being the Thirty-third of the Third Series. Edited by* N. DAVIES-COLLEY, M.A., M.C., *and* W. HALE WHITE, M.D. *With Plates and Woodcuts. Price to Subscribers, 6s.; to Non-subscribers, 10s. 6d. Postage free.*

---

CONTENTS.


1. Observations on the Treatment of Chlorosis by Hydrochloric Acid. By W. Hale White, M.D.
2. On some Interesting Pathological Conditions met with in Animals from the Post-mortem Room of the Zoological Gardens. By H. J. Campbell, M.D.
3. On the Localisation of Phthisis to the Apex of the Lung. By H. J. Campbell, M.D.
4. The True Lines of Advance in Anæsthetics. By J. Frederick W. Silk, M.D.
5. Some Points in the Morbid Anatomy of Leprosy. By Beaven Rake, M.D. (Communicated by the Editors.)
6. A Theory of Smallpox. By R. A. Birdwood, M.D. (Communicated by J. W. Washbourn, M.D.)
7. Seven Cases of Intra-spinal Hæmorrhage (Hæmatomyelia). By Alfred Parkin, M.S., M.D.
8. An Examination of Fifty Cases of Malignant Disease of the Stomach. By E. C. Perry, M.D., and L. E. Shaw, M.D.
9. A Case of Pemphigus affecting the Conjunctiva. By W. A. Brailey.
10. On Malignant Endocarditis. With Cases. By Frederick Taylor, M.D.
11. A Case of Ileo-colic Implantation, followed by Resection of Portions of the Large and Small Intestine for the Cure of Artificial Anus, due to Gangrene after Strangulated Hernia. By N. Davies-Colley.
12. A Research on the Properties of Pure Human Succus Entericus, with a few Notes on Absorption by the Bowel and Intra-abdominal Pressure. By A. H. Tubby, M.S., and T. D. Manning (from the Physiological Laboratory).
13. On the Volumetric Determination of Uric Acid in Urine. By F. Gowland Hopkins, B.Sc., F.I.C., Gull Research Student.
14. Note on a Form of Blood-pressure Manometer. By Ernest H. Starling, M.D., and W. M. Bayliss, B.A., B.Sc.
15. A Case of Actinomycosis of the Liver and Lungs. By Frederick Taylor, M.D.
16. List of Specimens added to the Pathological Museum during the year 1891. By Lauriston E. Shaw, M.D.

List of Pupils who have passed the Examinations of the several Universities, Colleges, &c., 1890.

List of Prizemen and Medallists, 1889–90.

List of Pupils who have received Appointments at Guy's Hospital, 1890.

List of Pupils who have passed the Examinations of the several Universities, Colleges, &c., 1891.

List of Prizemen and Medallists, 1890–91.

List of Pupils who have received Appointments at Guy's Hospital, 1891.


---

J. & A. CHURCHILL, New Burlington Street.

BINDING LIST FEB 15 1949

RC
31
L8G9
ser.3
v.34

Biological
& Medical
Serials

Guy's Hospital, London
Reports

PLEASE DO NOT REMOVE
CARDS OR SLIPS FROM THIS POCKET

UNIVERSITY OF TORONTO LIBRARY

STORAGE